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ChemRisk

Expert Report of Dr. Brent L. Finley

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Elnora Carthan, et al., vs. Rick Snyder, et al.

(Case Number: 5:16-cv-10444-JEL-EAS)

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February 3, 2023

I. EXPERIENCE

I am a board-certified toxicologist with over 30 years of experience conducting and managing studies involving chemical exposure and human health risk assessment. I have a bachelor's degree in Biological Sciences from Cornell University and a Ph.D. in Pharmacology/Toxicology from Washington State University. I am a principal at the ChemRisk Division of Stantec, a consulting firm providing state-of-the-art toxicology, industrial hygiene, epidemiology, and risk assessment services to organizations that face public health, occupational health, and environmental challenges. Over the last 25 years, I have authored over 400 health risk assessments related to the presence of chemicals in consumer products, foods, the environment, the workplace, households, and other settings. I have published over 160 peer-reviewed articles describing the health risk assessment of dioxins, polychlorinated biphenyls (PCBs), chromium, chlorinated solvents, and latex allergens. I have taught risk assessment courses at universities, have given numerous invited lectures, and have served on several risk assessment expert panels. I have been deposed and testified in state and federal court on matters related to chemical exposure and potential adverse effects in humans.

My curriculum vitae, which presents my background and training, is included as **Attachment A**.

II. MATERIALS REVIEWED IN FORMULATING OPINIONS

My opinions are provided in Section IV of this report. These opinions are based on my professional qualifications, work experiences, and knowledge of industrial hygiene, toxicology, health risk assessment, and related fields. My opinions also are based on information that is related to this case. In the process of preparing this report, I have reviewed and relied upon the following case-specific documents:

1. Second Amended Opinion and Order Granting in Part and Denying In Part Class Plaintiffs' Motion (dated August 31, 2021)
2. Corrected Order Regarding Certified Class Issues (dated November 2, 2022)
3. Fourth Consolidated Amended Class Complaint for Injunctive and Declaratory Relief, Money Damages, and Jury Demand (dated October 5, 2018)
4. Various medical records (various dates)
5. Deposition Transcript of Rhonda Kelso (dated November 12, 2019)
6. Continued Deposition Transcript of Rhonda Kelso (dated November 12, 2019)
    a. Various exhibits (various dates)
7. Deposition Transcript of Barbara Davis (dated November 13, 2019)
    a. Various exhibits (various dates)
8. Deposition Transcript of Darrell Davis (dated November 12, 2019)
    a. Various exhibits (various dates)
9. Expert Report of Clifford P. Weisel, MS, PhD (dated October 14, 2022)
    a. Various exhibits (various dates)
10. Expert Report of Daryn Reicherter, MD (dated October 18, 2022)
    a. CV (no date provided)
11. Expert Report of Howard Hu, MD, MPH, ScD (dated October 18, 2022)
    a. Various exhibits (various dates)

12. Expert Report of Robert A. Michaels, PhD, CEP (dated October 18, 2022)
    a. Various exhibits (various dates)
13. Expert Report of Panagiotis (Panos) G. Georgopoulos, MS, PhD (dated October 18, 2022)
    a. Various exhibits (various dates)
14. Various Files Related to Dr. Panos Georgopoulos's Modeling Simulations (various dates)
15. ASTI Home Inspection Report – 1910 Montclair Ave (dated January 4, 2021)
16. Revised ASTI Home Inspection Report – 119 Grace Street (dated January 5, 2021)
17. Remote Videotaped Deposition of Marc Edwards, PhD – Volume I (dated August 7, 2020)
18. Remote Videotaped Deposition of Marc Edwards, PhD – Volume II (dated August 10, 2020)
19. Remote Videotaped Deposition of Marc Edwards, PhD – Volume III (dated August 11, 2020)
20. Remote Videotaped Deposition of Panagiotis Georgopoulos (dated November 23, 2022)
    a. Various exhibits (various dates)
21. Remote Videotaped Deposition of Howard Hu, MD, MPD, ScD (dated November 21, 2022)
    a. Various exhibits (various dates)
22. Remote Videotaped Deposition of Robert A. Michaels, PhD, CEP (dated November 22, 2022)
    a. Various exhibits (various dates)
23. Dr. Robert Michaels. Lead (Pb) in Flint, Michigan Municipal Water v. Health Issues of Levy, Konigsberg Bellwether Plaintiffs and Potential Additional Plaintiffs (dated August 6, 2020)
24. Unified Coordination Group (UCG) – Flint Rash Investigation (dated August 2016)

In addition, I have reviewed and relied upon published papers and textbooks on toxicology and exposure assessment topics related to lead. Specific references cited in my opinions are listed at the end of this report. My opinions are expressed to a reasonable degree of scientific certainty. My time spent in the preparation of this opinion letter and reviewing documents to formulate my opinions as well as for any deposition or trial testimony I may be called upon to give will be billed at a rate of $550 per hour. These opinions are based on information available to me on the date of this report. I have provided a list of documents that were reviewed in forming my opinions. This report may be supplemented if new information becomes available. Where required, I will provide my demonstrative aides once they have been finalized.

III. CASE SPECIFICS

Background

On April 25, 2014, the City of Flint switched from water purchased from the Detroit Water and Sewerage Department (DWSD) to Flint River water as the source of tap water for Flint residents. Eighteen months later, on October 16, 2015, the City of Flint returned to the DWSD as their water source. Approximately 10 months into the "switchover period," Veolia North America (VNA) was retained by the City of Flint for consulting services over a 4-week period in February/March 2015.

The court certified a "Multi-Defendant Issues Class" on behalf of "[a]ll persons...who, for any period of time between February 10, 2015 and October 16, 2015, were exposed to…drinking water supplied by the City of Flint". "Exposure" is defined to include ingestion (either through drinking or consuming foods prepared with the drinking water), bodily contact with the water (such as by way

of bathing).” “Persons” is defined to include only those individuals who have reached the age of majority (18 years old) as of the date of the class notice (August 17, 2022).

I will be addressing the multi-defendant class in this report. In particular, I will be addressing the question defined by the court as “Issue 3: Were the contaminated water conditions capable of causing harm to Flint residents, properties, property and businesses?” for the period of February 10, 2015 to October 16, 2015.

My role in this case

In this report I rely on toxicology and health risk assessment principles to address issues related to the “multidefendant class”. Specifically, I describe the significant variability in the factors that influence lead exposure in adults and adolescents. I provide several specific examples of the variability in these factors within the general Flint population and certain defined groups within Flint, including class representatives [redacted] (K.E.K.), Ms. Rhonda Kelso, Mr. Darrell Davis, and Ms. Barbara Davis. I also discuss the validity of the assumptions underlying class plaintiff’s experts’ multidefendant class criteria, including a discussion of whether these criteria accurately identify Flint adult residents who may have experienced adverse effects from consumption of lead-containing tap water during the switchover period. I evaluate the entire switchover period, with an emphasis on February 10, 2015, to October 16, 2015 (the latter half of the period, which is the period identified in the definition of the “multi-defendant issues class”). I also evaluate the clinical evidence for the class representatives in terms of blood lead measurements collected in 2015 and 2016.

As part of my opinions, I discuss the work of the following plaintiff experts: Dr. Clifford Weisel, Dr. Panos Georgopoulos, Dr. Howard Hu, and Dr. Robert Michaels. Dr. Weisel developed a range of water lead levels (WLLs) that he maintains are representative of Flint tap water lead concentrations throughout the switchover period, including February 10, 2015 to October 16, 2015. Dr. Georgopoulos used a biokinetic lead model (the “AALM-Leggett model”) to estimate “’baseline blood lead level[s]’” and “likely” blood lead levels (BLLs) for different age groups hypothetically exposed to the WLLs provided by Dr. Weisel. I refer to this as the “hypothetical BLL framework”. Dr. Hu opined on the potential adverse effects associated with the estimated range of BLLs provided in the hypothetical BLL framework. In a separate opinion, Dr. Michaels opined on the potential induction of skin rashes and hair loss in Flint residents who may have used tap water during the switchover; these claims are related to water quality parameters and are unrelated to BLLs.

IV. OVERVIEW OF OPINIONS

Background on the health risk assessment methodology as it relates to this case

As noted above, I will be addressing the question defined by the court as “Issue 3: Were the contaminated water conditions capable of causing harm to Flint residents, properties, property and businesses?”.

To answer Issue #3, one must evaluate the relevant facts using a human health risk assessment methodology. Federal guidance for proper conduct of a health risk assessment has been in place

since the 1980's, and requires consideration of the following:

1) What was the intensity of the chemical exposure (dose)?
2) For how long did the exposure occur (duration)?
3) Was the chemical dose/duration sufficiently elevated to cause a particular health effect?

For the purposes of assessing the potential health risks associated with residential consumption of lead in tap water, measured and/or estimated blood lead levels (BLLs) are the metric of dose intensity, while the exposure duration is defined as a period of approximately 8 months (February 10, 2015 to October 16, 2015).

The Flint lead dose/durations (BLLs over an 8-month period) must then be compared to the minimum (threshold) lead dose/durations (BLLs over time) required to cause the alleged health effects. The minimum threshold values are identified from other lead-exposed populations where the health effects in question were evaluated as a function of lead dose/duration. If the Flint lead dose/durations are equivalent to or greater than the threshold dose/durations, then the answer to Issue #3 may be "yes"; if they are not, the answer is "no".

Dr. Hu agreed that the BLL is the proper metric of lead dose. He also agreed that a minimum lead dose (reflected by BLL) must be maintained over a minimum amount of time (duration) in order for there to be a risk of potential adverse effects.

However, as described in this report, no evidence is offered by Dr. Hu or any other plaintiff expert to indicate whether the threshold lead dose/durations for any alleged health effect were exceeded by Flint residents. Specifically, they did not attempt to demonstrate that any estimated or measured Flint adult BLL or water quality parameters maintained over an 8-month period, could have resulted in any of the adverse effects discussed by Dr. Hu or Dr. Michaels. In short, plaintiff experts did not answer the question posed by Issue #3, i.e., they did not present an analysis that proves the water conditions were capable of causing harm to any Flint resident.

As described in this report, the weight of evidence shows that the Flint adult BLLs were too low and the exposure period too brief for Flint residents to have exceeded the minimum dose/duration thresholds for any health effect alleged by plaintiff. Issue #3 should be answered as follows: The weight of evidence indicates that the water conditions were *not* capable of causing harm to Flint residents because the dose/durations were not sufficiently elevated to result in adverse effects".

My opinions are as follows:

- Opinion 1: The factors that influenced tap water lead dose/duration during the February 10, 2015 to October 16, 2015 period were highly individualized and subject to many variables that cannot be applied on a class-wide basis. As a result, lead dose/duration in Flint residents during the February 10, 2015 to October 16, 2015 period cannot be assessed on a class-wide basis.

- Opinion 2: The hypothetical BLL framework developed by plaintiff experts is based on several unfounded assumptions.
- Opinion 3: The hypothetical BLL framework developed by plaintiff experts contradicts the adult BLLs measured during the switchover period and does not accurately characterize tap water exposures to lead from February 10, 2015 to October 16, 2015.
- Opinion 4: Dr. Hu's claims of lead-related health effects that allegedly could have occurred due to tap water exposure from February 10, 2015 to October 16, 2015 are not supported by the scientific evidence.
- Opinion 5: Dr. Michaels' claims of skin rashes and hair loss due to tap water exposure from February 10, 2015 to October 16, 2015 are not supported by the scientific evidence.

Opinion 1: The factors that influenced tap water lead dose/duration during the February 10, 2015 to October 16, 2015 period were highly individualized and subject to many variables that cannot be applied on a class-wide basis. As a result, lead dose/duration in Flint residents during the February 10, 2015 to October 16, 2015 period cannot be assessed on a class-wide basis.

The court certified a "Multi-Defendant Issues Class" on behalf of "[a]ll persons and entities who, for any period of time between February 10, 2015 and October 16, 2015, were exposed to or purchased drinking water supplied by the City of Flint, owned real property in the City of Flint, or owned or operated a business in the City of Flint". It has been alleged that class members "have experienced and will continue to experience serious personal injury and property damage" as a result of exposure to "contaminated water".

As described in detail below, the members of the "class" as defined by plaintiffs have very little commonality regarding lead exposures that might have occurred during the February 10, 2015 to October 16, 2015 period. There are far too many exposure factors, each with a significant degree of variability and uncertainty, to permit consideration of the Flint adult population as a single "class" for purposes of assessing personal injury. Each Flint adult had their own unique lead exposure profile during the switchover period, and that profile will have very little commonality with most or all other Flint adults.

Furthermore, as described in this opinion and later in the report, tap water ingestion does not significantly influence a person's BLL relative to other exposure pathways such as the diet and soil and house dust ingestion. When developing their hypothetical BLL framework, plaintiff experts considered some aspects of variability in the tap water consumption pathway but not in the more important, more influential exposure pathways. This produces a narrative that falsely suggests small increases in tap water exposure will lead to a sustained, "quantifiable" increase in a person's BLL.

1.1 Tap water exposure variables

Class plaintiffs' experts acknowledge that tap water lead exposure is influenced by numerous factors

that have a significant degree of inter-individual variability. As described on pages 10-11 of Dr. Hu's report:

> **"**One challenge is that **"exposure" to water lead levels at residential taps** during and following the Flint Water Crisis **can be expected to vary spatially** (i.e. across locations within the city depending on the condition and type of service line, connectors, and indoor plumbing at each location an individual consumes tap water or item prepared with tap water); **temporally** (i.e. over time - the course of a day, week, and month, because of flushing, water flow, local pipe and interior plumbing conditions); **and frequency** of an individual's consumption of the tap water."

The following is also described on pages 11-12 of Dr. Weisel's report:

> "I will be identifying which potential sources of lead in tap water were affected by the corrosive water conditions due to the change in source of Flint's drinking water to the Flint River, thereby resulting in increasing the lead water concentration. Doing so presents several challenges since **tap water lead concentrations typically vary spatially** (i.e. across locations within the city **depending on the condition and type of service line**, connectors, and indoor plumbing at each location an individual consumes tap water or items prepared with tap water); **and temporally** (i.e. over time - the course of a day, week, and month, **because of flushing, water flow**, local pipe and interior plumbing conditions)."

Influence of service line composition on tap WLLs in Flint

As mentioned by Dr. Hu and Dr. Weisel, tap WLLs are highly influenced by the composition of public and private water service lines. Similar to other cities, lead pipes could be used for service lines in Flint until these pipes were banned in 1986 (Rabin, 2008). Galvanized service lines, which contained between 0.5 – 1.4% lead by weight until 2014 can also serve as a lead source (Clark et al., 2015; Pieper et al., 2018a; Pieper et al., 2018b; Pieper et al., 2017; Sandvig et al., 2008). Dr. Marc Edwards has testified that lead service lines are by far the most significant potential source of lead in tap water (Edwards Deposition Vol. III: p. 785, l. 8-9, 10, 18-19).

While lead mobilization can occur in lead and galvanized service lines, it does not readily occur in copper service lines. This was confirmed by the tap water sampling performed in Flint by Pieper et al. (2018a), a study relied upon by Dr. Weisel, that showed significantly lower lead concentrations in copper service lines compared to homes with lead, galvanized, or unknown service lines. Specifically, as shown below in Figure 1, Pieper et al. (2018a) noted that in August 2015, the median first draw WLLs were statistically significantly lower ($p < 0.05$) in homes with copper service lines (2.4 ppb) compared to homes with service lines made of lead (7.6 ppb), galvanized (5.8 ppb), or unknown material (6.3 ppb).

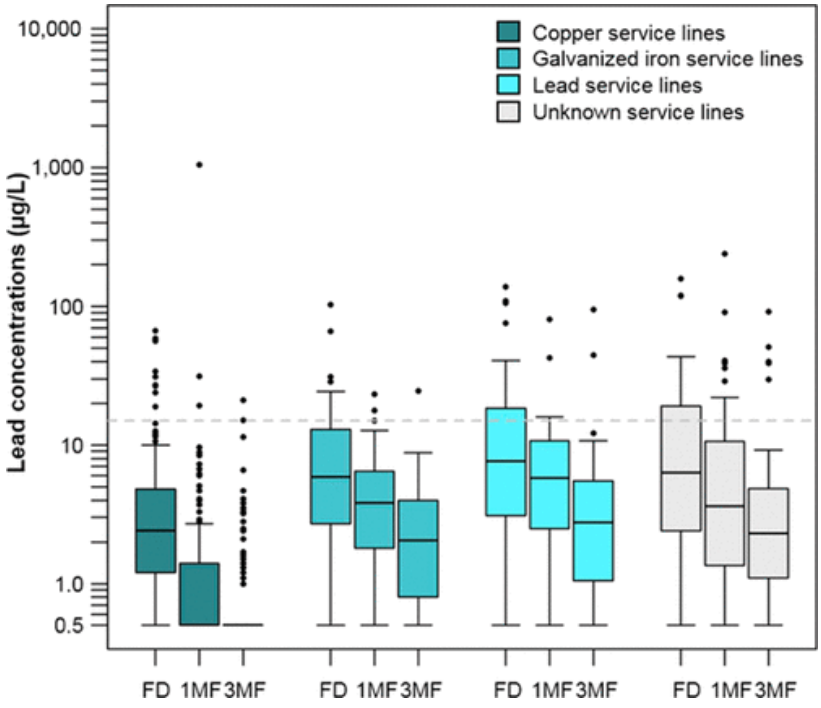


Figure 1. *Figure 1 from Pieper et al. (*(Pieper et al., 2018a)

Also, as shown in Figure 1 above, first draws from the tap had greater WLLs than subsequent draws, regardless of service line composition. First draws are a measure of the WLL after the water has remained stagnant in the plumbing for several hours (usually the first time the tap is used in the morning), while "flush" samples are generally representative of the WLLs throughout the remainder of the day. WLLs in first draws are typically higher than those in subsequent draws, as was observed by Pieper et al. (2018a) for each type of service line. This is another source of variability that can significantly influence WLLs even within a single type of service line. For homes with copper service lines, 115 of the 228 samples (>50%) did not have a detectable WLL at a limit of detection of 1 ppb; this was due to a high percentage of non-detect WLLs in the flush samples. As noted above, Dr. Weisel agreed that WLLs vary by service line composition.

In summary, tap WLLs throughout Flint were highly variable and statistically significantly different during the February 10, 2015 to October 16, 2015 period, depending on service line composition and whether the WLL was taken from a first draw or a subsequent flush sample. This factor alone raises serious questions regarding the merits of treating all adolescents and adults in Flint as a single class.

Many adult Flint residents had significantly reduced or eliminated their tap water consumption before the February 10, 2015 to October 16, 2015 period

To construct the hypothetical BLL framework, the plaintiff experts assumed that Flint residents consumed tap water, at the default consumption rate provided by the AALM model (approximately one liter of water consumed per day), for 90 consecutive days during the switchover period. The period of ninety consecutive days was chosen because that is the time frame after which BLLs reach

a steady state condition (i.e., they do not continue to increase significantly even with continued water consumption, as indicated by the AALM model).

It is critical to note that the BLL framework is inconsistent with the class definition. As mentioned earlier, the multi-defendant class is defined as "[a]ll persons ...who, **for any period of time** between February 10, 2015 and October 16, 2015, were exposed to … drinking water supplied by the City of Flint". "[F]or any period of time" obviously would include consumption periods for far less than 90 days. Hence, plaintiff experts ignored the variability inherent in tap water consumption rates as contained in the class definition. The BLL framework in fact only represents a "worst-case" analysis of tap water lead exposure that is unlikely to apply to more than a few (if any) Flint residents.

Many Flint residents began to complain of changes to the water quality shortly after the switch to the Flint River as the tap water source in April 2014. To varying degrees, Flint residents minimized or completely discontinued their use of tap water for drinking, cooking, bathing, washing dishes, and other uses during the first half of the switchover period. Published data demonstrates that Flint residents consumed much less tap water in 2015. For example, Christensen et al. (2019) reported a large, statistically significant, and sustained increase in sales of bottled water in Genesee County corresponding with the issuance of the 2014 boil advisories. Gomez et al. (2019) reported an overall decline of residential water usage in Flint from 2013 to 2015, in which the average per user declined from 290 centrum cubic feet (CCF) in 2013 to 97 CCF in 2015; this represents a 67% decrease in water usage.

In fact, there is testimony indicating that the class representatives reduced their tap water ingestion after the water source was switched in April of 2014:

Darrell Davis

[redacted]

Barbara Davis

[redacted]

Rhonda Kelso

[redacted]

[redacted]

K.E.K.

[redacted]

Plaintiffs' expert Dr. Georgopoulos also acknowledged the decline in 2015 tap water consumption rates in Flint following the water source switchover:

> When asked by counsel if there was a decrease in the amount of tap water that was being used by Flint residents when the city switched to Flint water, Dr. Georgopoulos stated "[t]hat is correct" (Georgopoulos Depo 11/23/2022: p. 60, l. 11).
>
> Specifically, Dr. Georgopoulos stated that the "use of unfiltered tap water was reduced dramatically after January 2015" (Georgopoulos Depo 11/23/2022: p. 59, l. 15-17).
>
> Dr. Georgopoulos also confirmed when asked by counsel that the reduction in water use by Flint residents was up to 70% after the switchover (Georgopoulos Depo 11/23/2022: p. 60-61).
>
> When asked by counsel about the decrease in BLLs in Flint female adults reported in the Gomez et al. (2019) paper, which has a study period of April 2014 to October 2015, Dr. Georgopoulos stated "that th[e authors] include ten months where the consumption of tap water had been decreased by 70 percent. So…of course you would expect the blood levels to go down" (Georgopoulos Depo 11/23/2022: p. 178-179).

Although significant decreases in tap water consumption rates during the February 10, 2015 to October 16, 2015 period were acknowledged by plaintiff experts, no attempt was made to consider this fact when predicting BLLs in this case.

Variability in tap WLLs at any given time point

As discussed above, Flint tap WLLs are significantly influenced by service line composition and the presence/absence of water filters. For lead-containing service lines, WLLs also vary by stagnation time, pipe length, and many other factors.

Because these factors varied across Flint, one would expect tap water WLLs across Flint to be highly variable at any given time point and sampling efforts during the switchover have proven that to be the case. Sampling from the 2015 Flint Water Study found WLLs ranging from non-detect (ND) to 500 ppb across the City of Flint (Figure 2). The great majority of the samples were below the EPA LCR action level of 15 ppb. Dr. Weisel also agreed that WLLs can vary temporally. In his report, he stated:

> "The lead concentration in drinking water varies during a single day and across days within a single household … Further, the water lead levels in homes which had multiple samples collected over several hours in a single day, both during and after the crisis period were highly variable" (Weisel Report 10/14/2022: p. 21)

> "Water samples collected at different times after a tap is first opened can have different lead levels dependent upon how long, the flow rate, and what portion of the plumbing the water was in contact with in homes and larger buildings, such as schools and office buildings … When sequential water samples were taken in Flint homes after water corrosion protocols were put back into place, elevated and variable lead water levels were still found" (Weisel Report 10/14/2022: p. 22-23)

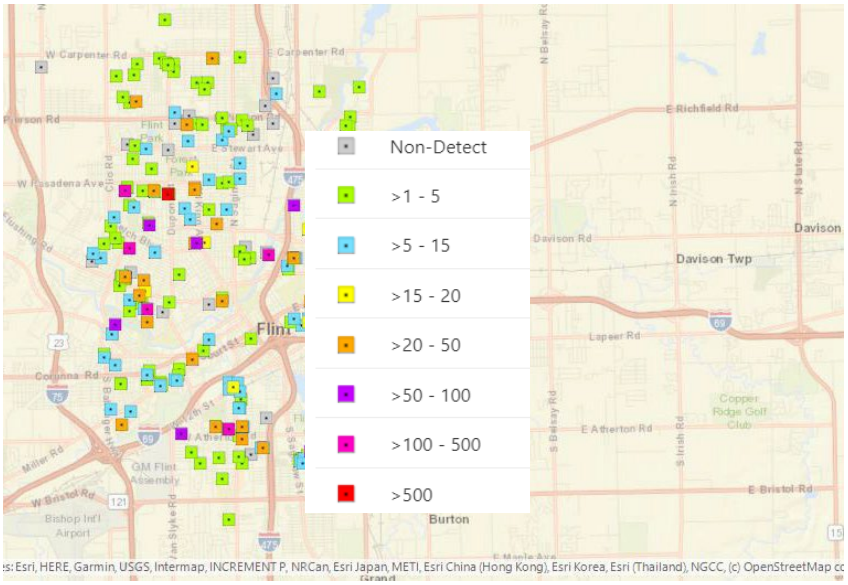


Figure 2. *2015 sampling results from the Flint Water Study.*

Figure 3 shows the locations of 4,051 water sampling results taken in Flint in January 2016. Again, the vast majority of homes had WLLs below the EPA action level of 15 ppb, and over half were non-detect. Only 6.3% (or 255) of the homes tested had water lead levels above the action level. More importantly however, WLLs varied greatly across the city, with no apparent spatial pattern.

Dark green: 0 ppb - **52.7%**
Light Green: 1-4 ppb - **30.5%**
Yellow: 5-14 ppb - **10.4%**
Orange: 15-49 ppb - **4.2%**
Red: 50-149 ppb - **1.3%**
Purple: 150 and above - **0.8%**

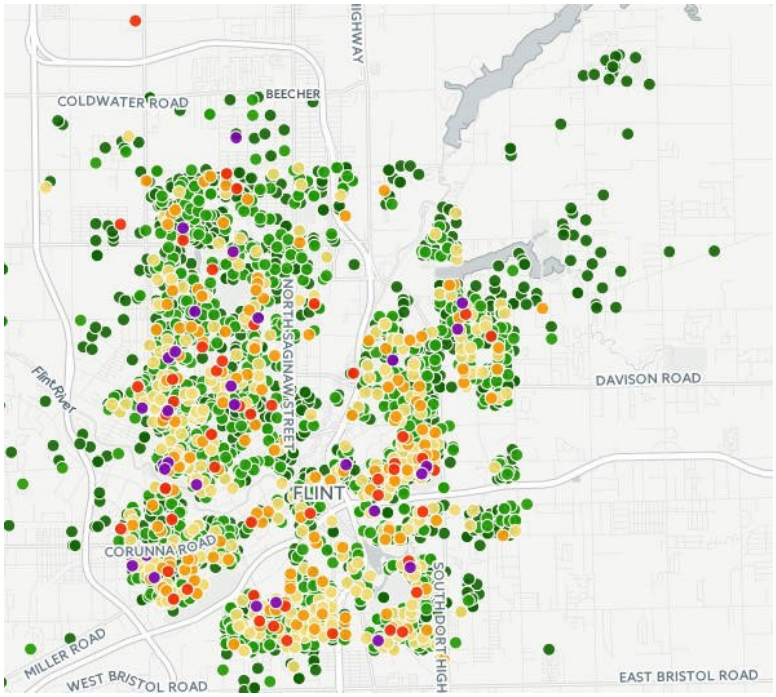


Figure 3. *Map of WLL sampling (n=4,051) taken during January 2016*

Summary: variability for tap water exposure factors

There is a significant degree of variability present in every factor that influences the tap water exposure pathway. This degree of variability indicates that Flint adults cannot be treated as a single group of individuals.

1.2 Non-tap water exposure variables

In addition to the variability inherent in every tap water exposure assumption, non-tap water exposure pathways also contain significant inter-individual variability, as described below.

Dietary intake of lead

In adults, diet is the main source of an individual's lead exposure and is the primary contributor to the BLL. The fact that dietary lead is ubiquitous and serves as the largest source of an adult's lead exposure has been recognized by both governmental authorities, as well as the peer-reviewed literature.

Lead is ubiquitous in various food products either via direct absorption from soil (fruits, vegetables, and grains), aerial deposition plant surfaces, or via different manufacturing processes during which foods come in contact with lead-containing surfaces (FDA, 2020). As shown in Figure 4, the lead contents of different foods vary considerably, and many common foods contain lead concentrations far higher than the average WLLs measured in Flint tap water during the switchover period.

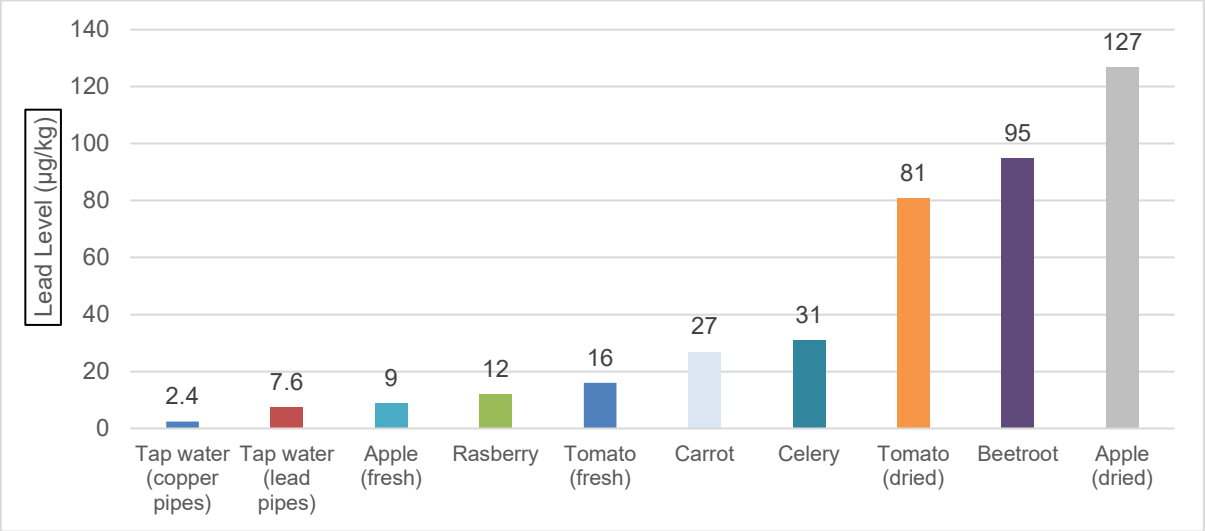


Figure 4. *Lead concentrations of various common foods vs. Flint tap WLLs from different service line compositions as reported by Rusin et al. (2021) and median of first draw 2015 WLLs from Pieper et al. (2018a), respectively. The WLLs for tap water were converted from µg/L to µg/kg using a density value for water of 1.0 kg/L.*

The significant contribution of diet to adult BLLs can be illustrated via the AALM model used by plaintiff experts. As shown in Table 1 below, using the same age-specific default exposure parameters in the AALM that were used by plaintiffs' expert Dr. Georgopoulos, diet contributes the most to total daily lead exposure (Table 1).

Table 1. *Contribution of various lead exposure pathways to total daily lead exposure (Females at 1 ppb WLL).*

Age (year)	Air (µg/day)	House Dust (µg/day)	Soil (µg/day)	Water (µg/day)	Diet (µg/day)	TOTAL (µg/day)
10	1.5	1.6	1.6	0.5	5.0	10.2
15	1.7	0.8	0.8	0.5	10.0	13.9
20	1.8	0.8	0.8	0.6	11.0	15.0
30	1.9	0.8	0.8	0.7	13.0	17.2
40	1.9	0.8	0.8	0.9	14.0	18.4
50	1.9	0.8	0.8	1.0	15.0	19.5
60	1.9	0.8	0.8	1.0	15.0	19.5
70	1.9	0.8	0.8	1.0	15.0	19.5
80	1.9	0.8	0.8	1.0	15.0	19.5
90	1.9	0.8	0.8	1.0	15.0	19.5

Specifically, in every age group, the diet is associated with the highest daily lead dose. Also, variability in the dietary exposure pathway across the different age groups is greater than that of the tap water ingestion pathway.

As shown in Figure 5 below, for the 60-year-old female group, dietary lead contributes to approximately 77% of total daily lead exposure (Figure 5).

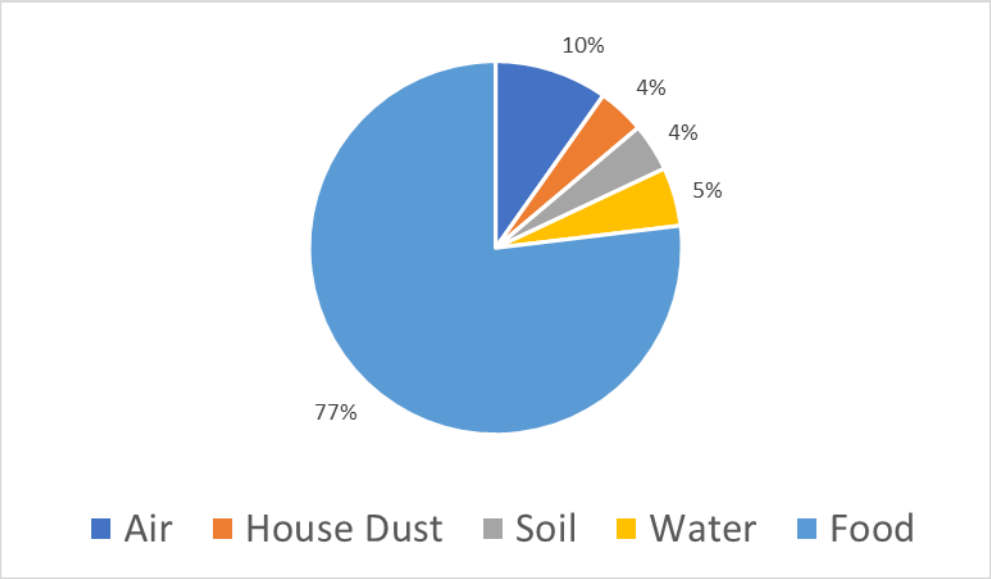


Figure 5. *Percent contributions to daily lead exposure by source (µg/day) for a 60-year-old female using the same age-specific exposure parameters as those used by plaintiff experts*

Variability in dietary lead exposure intake was not accounted for in the plaintiff experts' analysis and instead, this exposure pathway was simply treated as a single, unvarying point estimate for each age group.

Lead-containing paint and house dust

Flaking and chipping of lead-based paint (LBP) contributes to exterior contamination of soils surrounding the home and to lead dusts within the home. Lead in residential paint was banned in 1978, but over 90% of the homes in Flint were built prior to 1980, and the vast majority were built prior to 1960 (Figure 6) (Abernethy et al., 2018; U.S. Census Bureau, 2018).

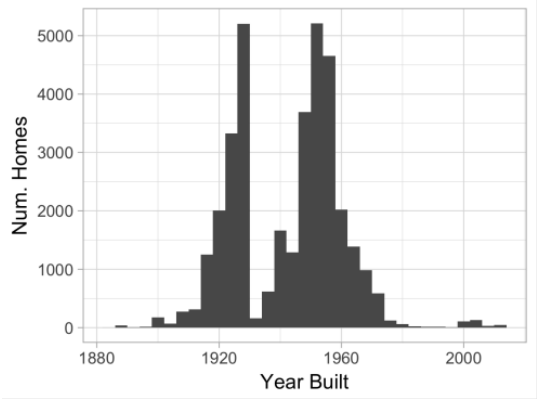


Figure 6. *Distribution of the houses in Flint and the year that they were built (Abernethy et al., 2018)*

The fact that many Flint adults lived in homes with lead-containing paint, while others did not, means that the influence of a significant lead source varied greatly across the adult population of Flint; i.e. there is variability between the lead exposure profile of a Flint adult who lived in an LBP-containing home vs. one who did not. Further, there is significant variability even within the subset of adults living in LBP homes. Variable factors for any given LBP home include: the amount of lead in the paint that was used, the degree of chipping and flaking and overall condition of painted surfaces, housekeeping practices (i.e., ambient dust load), whether and how many times a home was repainted, and the degree of contact an adult has with interior house dusts (e.g. presence/absence of door mats and interior carpeting, access to window wells).

Class representatives K.E.K. and Rhonda Kelso resided in a home ( ) that had Class representatives Barbara and Darrell Davis also resided in a home . Yet, plaintiff experts made no attempt to account for the variability introduced by the presence of lead-based paint. Instead, the lead level in house dust was treated as a single, unvarying point value that only represented homes without lead-based paint (80 ppm).

Lead-containing soil

Laidlaw et al. (2016) extracted data from the Edible Flint local food collaborative to generate a map of the estimated soil lead concentrations in Flint by census tract. The surface soil lead concentrations from garden soils and other locations are shown in Figure 7 below. As can be seen in the figure, soil lead levels are relatively higher in the city interior. This is consistent with studies of other inner cities and is a "result of a higher concentration of lead sources in the higher density… cores of older cities" specifically that have "significant historic inputs from…industry" (Laidlaw et al., 2016: p. 8).

It is clear from Figure 7 that much of Flint has soil lead levels that far exceed the mean background level of 17 ppm in Michigan (USGS, 2010). Furthermore, some areas of Flint have soil lead levels that exceed the EPA health-based value of 200 ppm, including as discussed below, the properties of the class representatives. It is important to note that, because the Edible Flint program targeted garden soils, which would not be influenced by exterior chipping and flaking of lead-based paint, the soil lead levels reported in Laidlaw et al. (2016) are biased low relative to yard soils that were impacted by lead-based paint.

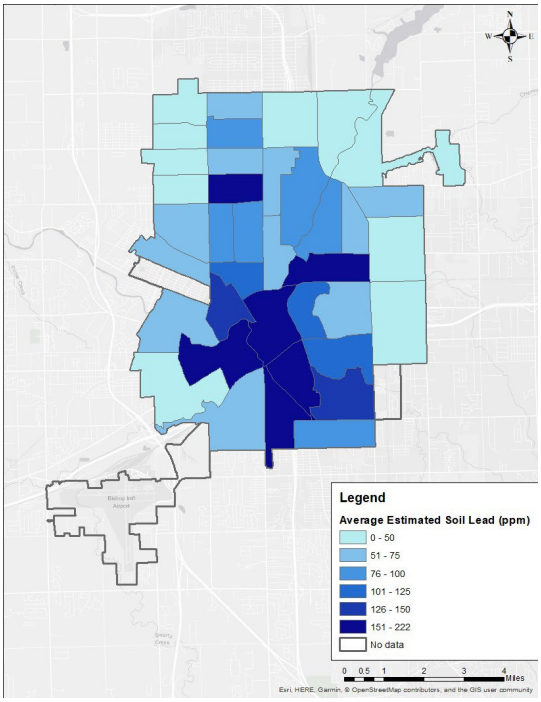


Figure 7. *Average estimated soil lead concentration by census tract in Flint, Michigan. The soil lead was derived from extractable lead concentrations via a linear regression model. Adapted from Figure 4 of Laidlaw et al. (2016).*

Finally, it is clear from Figure 7 that the soil lead concentrations in Flint are highly variable.

Variability in soil lead concentrations is also apparent for the plaintiffs in this case as shown in Table 2 for class plaintiffs K.E.K., Rhonda Kelso, Barbara Davis, and Darrell Davis (ASTI Davis 1/4/2021; ASTI Kelso 1/5/2021). From these data, it is clear that soil lead can greatly vary, even at a single residence.

Table 2. *Soil lead sampling results for class plaintiffs K.E.K., Rhonda Kelso, Barbara Davis, and Darrell Davis*

	Sample Number	Location	Lead (mg/kg)
K.E.K and Rhonda Kelso ([redacted])	SL-01	C/D Corner	[redacted]
	SL-02	Rear Yard	[redacted]
	SL-03	Side B, Dripline	[redacted]
	SL-04	Side A, Dripline	[redacted]
Barbara and Darrell Davis ([redacted])	SL-01	Side A, Yard	[redacted]
	SL-02	Side B, Dripline	[redacted]
	SL-03	Side C, Yard	[redacted]
	SL-04	Front Porch	[redacted]

Despite this, plaintiff experts made no attempt to account for the variability in soil lead levels and instead soil lead was treated as a single, unvarying point value of 100 ppm.

Other non-tap water lead sources

In addition to diet, soil, and house dust, there are numerous other potential sources of lead in a household including:

- Ceramic pottery, glassware, and cookware
- Spices from international origins
- Dietary supplements and homeopathic remedies
- Cosmetics and religious powders
- Amulets, charms and jewelry
- Track-home lead from occupational exposure

The degree to which these other sources will contribute to person's BLL is a highly individualized and variable factor.

Nutritional status affects lead absorption following WLL exposures and this factor is highly variable

The extent and rate of gastrointestinal absorption of ingested inorganic lead are influenced by physiological parameters such as fasting and nutritional calcium and iron status (ATSDR, 2020).

- Fasting status

The presence of food in the gastrointestinal tract has been shown to decrease absorption of water-soluble lead in adults (Blake and Mann, 1983; Blake et al., 1983; Heard and Chamberlain, 1982; James et al., 1985). Specifically, the absorption of a tracer dose of lead acetate in water was approximately 63% when ingested by fasted adult subjects versus 3% when ingested with a meal James et al. (1985); nearly identical results were reported by Heard and Chamberlain (1982). Additionally, these studies also observed that mineral content is one contributing factor to the lower absorption of lead when lead is ingested with a meal; the presence of calcium and phosphate in a meal has been shown to depress the absorption of ingested lead (Blake and Mann, 1983; Blake et al., 1983; Heard and Chamberlain, 1982)

Plaintiff experts made no attempt to account for the variability introduced by fasting status and lead absorption and instead, lead absorption was treated as a single, unvarying point value.

<u>Summary: Sources of variability in lead exposure pathways</u>

Table 3 summarizes the numerous exposure factors that are highly variable.

Table 3. *Summary table of exposure variables that influence the lead exposure profile of an individual.*

Summary of Variable Factors in Lead Exposure	
Sources of variability in tap water exposure pathways	Service line composition (copper, lead, galvanized)
	Exposure Duration
	Exposure Frequency
	General variance in WLLs
	Stagnancy
	Water Filter Use
Sources of variability in non-tap water exposure pathways	Exposure to lead through diet
	Exposure to lead-containing paint and house dust
	Exposure to lead-containing soil
	Exposure to other various lead-containing objects
	Biological factors (e.g., nutritional status, fasting)

Most of these exposure factors directly influence an individual's daily lead dose, and therefore it is inevitable that there was significant inter-individual variability in daily lead dose during the February 10, 2015 to October 16, 2015 period. Exposure duration also has a significant degree of inter-individual variability because, although the designated "exposure period" is approximately 8 months, residents consumed tap water for varying lengths of time during that period (and as noted above, none of the class representatives consumed the tap water at all during that period).

Plaintiff's failure to consider variability in the numerous exposure factors that dictate a person's BLL leads to a false conclusion that small increases in WLL exposure will result in a sustained increase in a person's BLL

The alleged "increased" BLLs in plaintiff's hypothetical BLL framework are simply an artifact of treating a single exposure pathway (tap water ingestion) as a variable while ignoring the variability in all the other exposure pathways. Plaintiff's methodology more accurately represents a sensitivity analysis, which is merely a tool used to evaluate the degree to which a model output changes in response to changes in specific input assumptions. The output of a sensitivity analysis is not intended to be used to assess exposure via all pathways combined, nor is it intended to be used to reach health risk conclusions. This is explored further in Opinion #3.

Opinion 2: The hypothetical BLL framework developed by class plaintiffs' experts is based on several unfounded assumptions.

To construct the hypothetical BLL framework, the plaintiff experts assumed that Flint adults consumed tap water, at the default consumption rate provided by the AALM model, for at least 90 consecutive days during the switchover period.

Background information on the AALM model

The AALM is an outgrowth of the Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in

Children. Version 0.99d of the IEUBK was released in 1994 and has been widely accepted in the risk assessment community as a tool for implementing the site-specific risk assessment process when the issue is childhood lead exposure. The IEUBK model was designed to assess changes in blood lead of children over periods of no less than three months (pseudo steady-state conditions).

Over time, the need for lead modeling for individuals other than children and/or varied exposure durations provided the impetus to develop a more versatile biokinetic model for lead. As a result, EPA's Office of Research and Development (ORD) in collaboration with the Office of Chemical Safety and Pollution Prevention (OCSPP) developed the AALM to provide a tool for evaluating the impact of possible sources of lead on blood and other tissue levels (including bone) in humans from birth to 90 years of age for intermittent lead exposures of a day or more as well as stable exposure conditions. Compared to the IEUBK Lead Model for Children, the internal kinetics of the AALM are more transparent and accessible to the user. The current External Review Draft of the AALM (Version 2.0, 2019) reflects updates from a previous 2005 draft and input from EPA's Science Advisory Board (SAB).

The most recent EPA SAB review (8/20/2022) of the AALM recommended numerous modifications and clarifications to the model. As such, the present version (2.0) is still undergoing beta-testing and has not been fully validated by the EPA for regulatory applications. A particular concern in establishing AALM input parameters is evidenced in the relative bioavailability (RBA) for soil-borne lead. Most of the research on lead absorption from soil has focused on young children (who represent a more highly exposed, sensitive sub-population) and adults (mainly from occupational exposure). There is a paucity of data on the absorption profiles of adolescents (ages 8 -18). The AALM is left to interpolate RBA values between young children (38%) and adults (12%) for the adolescent age range.

The SAB review (P.5) also discussed the lack of a statistical module for model output: "Each simulation generates a single time series of predicted mean blood lead concentrations over time, summarized in both an Excel table and graphic format. By contrast, the IEUBK and Adult Lead Models generate probability distributions of blood lead concentrations by applying a lognormal distribution model to the predicted mean concentrations. This utility does not currently exist in the AALM and it is not clear if the AALM outputs represent geometric mean values, though if they are users could post-process the results on their own." The practical implication of this limitation is that point estimate exposure parameters yield a singular point estimate blood lead concentration. The inherent variabilities in exposure and biokinetic parameters are not captured, thus denying the user of valuable information on the probability that actual blood lead concentration may be higher or lower than the predicted point estimate value. Such a limitation has the effect of imparting on the AALM a degree of precision that empirical data on populations of similarly exposed individuals does not reflect.

<u>Unfounded assumptions used to develop the hypothetical BLL framework</u>

- *Incorrect assumption that WLLs were non-detect before the switchover*

According to the expert report of Dr. Georgopoulos, the assumption of a baseline WLL of 1 ppb is

based on his belief that this value was half of the minimum reportable level before the switchover, and that the lead and copper rule (LCR) compliance sampling performed by the City of Flint in the two water sampling periods prior to the switchover found that there were no Flint houses sampled that had a detectable WLL.

While it is true that the LCR compliance sampling from the Flint water system prior to the switchover period reported no detectable WLLs in the Flint tap water, it is also known (to plaintiff experts) that the instructions provided to residents as part of the compliance monitoring program yielded results that are biased low. In his June 24, 2015 memo, Del Toral (2015) from the USEPA pointed out that the practice of pre-flushing before collecting compliance samples minimized lead capture and underestimated WLLs. He expressed concerns that the instructions the City of Flint gave to residents to pre-flush based on MDEQ recommendations resulted in a serious underestimation of the WLLs in Flint water. Given that these recommendations were in place prior to the switchover period, it is clear that pre-switchover LCR compliance sampling also suffered from the same under-estimation issues.

Roy et al. (2019) made specific reference to the City of Flint LCR data relied upon by Dr. Georgopoulos, noting that it was "nearly useless" because the results were biased low due to 1) failure to meet the requirement that at least 50% of the samples be collected from homes with lead-containing service lines, and 2) pre-flushing of the tap lines before sample collection. Regarding the 50% minimum criterion, Dr. Edwards noted that, rather than target homes with lead-containing service lines, the sampling was "at best a random sampling and at worse was even selecting homes that had … low lead" (Edwards 8/10/20: p. 370, l. 13-16, 24 – p. 371, l. 1, 3-5). Pieper et al. (2018a) noted that the City of Flint continued to mismanage the LCR sampling throughout the switchover period.

In fact, detectable WLLs were present in tap water in homes throughout Flint for years before the switchover. Figure 8 below provides ample evidence that tap WLLs in Flint were not <1 ppb prior to the switchover.

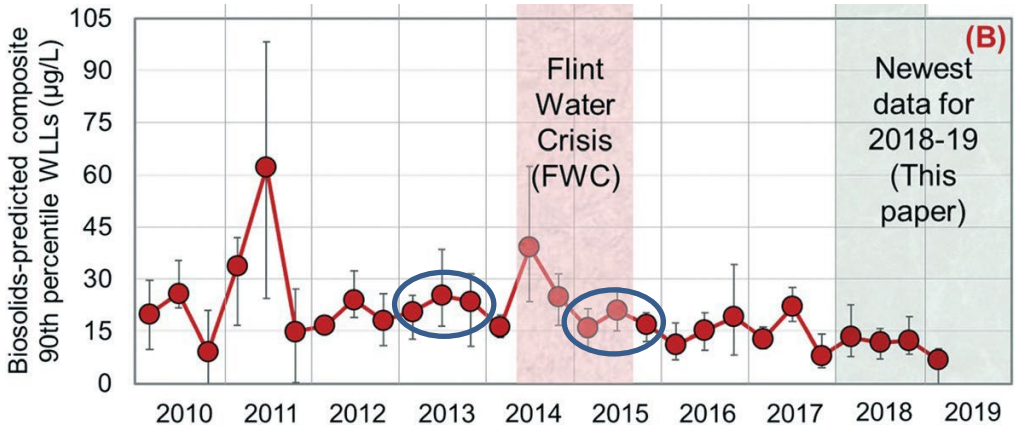


Figure 8. *Estimated Flint tap WLLs before, during, and after the switchover period. Figure 1B from (Roy and Edwards, 2020b).*

In summary, the assumption by plaintiff's experts that WLLs were below 1 ppb prior to the switchover period creates a misleading and self-serving narrative that the Flint tap water was "pristine" and lead-free prior to the switchover and that any detectable WLL during the switchover is by definition an "increased WLL". The fact is that exposure to tap WLLs was occurring well before the switchover and as can be seen in Figure 8, the LCR action level of 15 ppb was exceeded on numerous occasions between 2010 and 2013. This undermines the validity of the hypothetical BLL framework.

- *Failure to consider the fact that tap WLLs during February 10, 2015 to October 16, 2015 were lower than pre-switchover WLLs*

Two recent studies have reported a comprehensive analysis of Flint WLLs before and during the switchover: (Roy et al., 2019) and Roy and Edwards (2020b). In Roy et al. (2019), the investigators found a strong temporal correlation between WLLs measured in tap water versus lead levels in biosolids collected at the Flint wastewater treatment plant. They used this relationship to estimate 90th percentile WLLs in Flint for several years prior to and throughout the switchover period based on the biosolids lead data that had been measured during those periods. The 2020 paper is simply an update that presents more recent data for 2018 and 2019 (well after the switchover period).

Prior to the switchover period, the Flint WLLs remained at a relatively steady "baseline" level throughout 2012 and 2013 until a WLL spike occurred in 2014. The timing of the 2014 WLL spike coincides with the switch to Flint River water. The switch to Flint River water resulted in the 2014 WLL spike. All wastewater sources (kitchen and bathroom taps, washing machines, flushed toilets, dishwashers, bathtub and shower drains, etc.) contributed to the daily mass of lead released in the wastewater by each residence. The 2014 WLL spike then subsided to a baseline level before the end of 2014, and the WLLs have remained at a slowly decreasing baseline level ever since (Roy and Edwards, 2020a).

The key finding relevant to this case is that the 2015 baseline WLLs, including the February 10, 2015 to October 16, 2015 period, were lower than the pre-switchover 2013 baseline WLLs. These papers were never discussed or even cited in any plaintiff expert report, suggesting that a less than thorough weight of evidence (WOE) review was conducted by plaintiff experts. The WOE in this case indicates Flint tap WLLs during February 10, 2015 to October 16, 2015 were lower than in 2013 (little if any weight can be assigned to the City of Flint LCR data, as discussed earlier). Obviously, since 2015 WLLs were lower than pre-switchover levels, tap water consumption during 2015 does not meet the plaintiffs' injury criterion of exposure to increased WLLs because the WLLs were not "increased" during February 10, 2015 to October 16, 2015.

- *Failure to acknowledge that most of the hypothetical WLLs used to construct the BLL framework are upper-bound values that have no relevance to a vast majority of Flint tap WLL exposures*

The hypothetical BLL framework was developed using WLLs that range from 1 ppb to 300 ppb. In Dr. Georgopoulos's declaration, he states that the 300 ppb concentration represents "a value from the upper tail" of the distributions of WLLs measured in Flint during the switchover period. In order to better understand how the range of WLLs used in this analysis is representative of WLLs that the

Flint population was exposed to, I evaluated the WLL database compiled by Dr. Goovaerts (samples collected by MDEQ and the Virginia Tech research team as part of the 2015 Flint Water Study). This combined database includes 932 WLL measurements with 288 (31%) of them being below the limit of detection of 1 ppb. The WLL percentiles from the data set associated with each hypothetical WLL are presented in Table 4. The median WLL (50th %ile) for this combined data set is approximately 2 ppb. Half of the WLLs used to construct the hypothetical BLL framework (20-300 ppb) are beyond the 90th percentile. In short, most of the WLLs used to develop the hypothetical BLL framework are far too high to be relevant to most (if any) Flint residents. The 93%ile value of 20 ppb in Table 4 closely matches the 2015 90%ile values of 15-20 ppb in Figure 8 from Roy et al. (2019).

Table 4. *WLL percentiles used to develop plaintiff's hypothetical BLL framework*

WLLs used to develop hypothetical BLL framework (ppb)	Percentile of Combined MDEQ and 2015 Flint Water Study Dataset
1	31%
2	45%
3	60%
5	73%
10	86%
20	93%
50	97.5%
100	99%
200	99.7%
300	99.85%

- *Failure to consider the fact that many adults significantly decreased their tap water consumption and/or did not consume tap water for 90 consecutive days during the switchover period*

To construct the hypothetical BLL framework, the plaintiff experts assumed that Flint adults consumed tap water, at the full default consumption rate (liters of water consumed per day) provided by the AALM model, for at least 90 consecutive days during the switchover period.

It is obvious that these assumptions would not apply to most Flint adults, particularly in the eight-month period between February 10, 2015 and October 16, 2015. Specifically, it is known that Flint adults decreased their tap water consumption before or during 2015, either by consuming the water intermittently (i.e., not daily) and/or by consuming the water at a reduced rate on those days when consumption did occur. As noted earlier in Opinion #1, Christensen et al. (2019) reported a significant, and sustained increase in sales of bottled water in Genesee County corresponding with the issuance of the 2014 boil advisories. Gomez et al. (2019) reported a significant decline of residential water consumption in Flint from 2013 to 2015. Dr. Georgopoulos acknowledged the likelihood of reduced consumption, and lower exposures, in his declaration:

> "Infrequent and non-continuous exposures (i.e., less than 1 day per week over a minimum duration of 90 days) would be expected to produce oscillations in BLL associated with the

absorption and subsequent clearance of BLL between each exposure event. Within the "persons" considered here, there will, of course, be gradations of lead exposure, with some individuals having lower exposures, and other individuals having higher exposures".

In fact, as noted earlier, Dr. Georgopoulos even acknowledged that reduced consumption led to reduced adult BLLs during the switchover period: when asked by counsel about the decrease in adult BLLs reported in the Gomez et al. (2019) paper (which occurred during the switchover period), Dr. Georgopoulos stated "that th[e authors] include ten months where the consumption of tap water had been decreased by 70 percent. So…of course you would expect the blood levels to go down" (Georgopoulos Depo 11/23/2022: p. 178-179). In other words, Dr. Georgopoulos specifically acknowledged that he *expected* adult BLLs would decrease during the switchover period due to reduced tap consumption, and then (contrary to his own testimony) proceeded to ignore that belief and assumed full daily consumption rates to develop the hypothetical framework of "increased BLLs" during the switchover period.

In short, due to the nature of the assumptions used to construct the BLL framework, the framework does not apply to most Flint adults during February 10, 2015, and October 16, 2015 and conflicts with the testimony of plaintiffs' expert.

- *Failure to consider factors that reduce systemic lead uptake from tap ingestion*

Dr. Georgopoulos relied on the single point estimate default value of 100% to estimate lead uptake following tap water ingestion. As noted earlier, the presence of food in the gastrointestinal tract significantly decreases absorption of water-soluble lead in adult individuals; values as low as 3% absorption have been reported. A value of 100% applies only to tap water consumed on an empty stomach. Therefore, plaintiff expert's assumption of 100% absorption of ingested WLL far over-estimates the average absorption rate lead in tap water and this leads to an overestimate of BLLs in the hypothetical BLL framework as well as an overestimate of % contribution of WLL to BLL.

- *Failure to consider the fact that almost all Flint adults live in homes with lead-based paint*

As discussed in Opinion #1, most homes in Flint contain lead-based paint. Lead-based paint is a major contributor to BLLs (far more than tap water) because the paint results in increased lead levels in indoor house dust. However, for reasons that are not explained, the indoor dust exposures estimated by Dr. Georgopoulos only considered the contribution of outdoor soil to indoor dust lead levels; any potential indoor dust exposures due to deteriorating lead-based paint or other indoor sources of lead were excluded. As a result, class plaintiff experts' methodology far over-estimates the percent contribution of tap water to the total lead exposure of any individual living in Flint during the switchover period. This is especially true for 2015, when tap water exposures were low.

Actual sampling of soil and indoor dust at class representative properties contradicts plaintiff experts' assumption that lead in indoor dust is solely from outdoor soil. For example, at the residences of Darrell and Barbara Davis ( ) and K.E.K. and Rhonda Kelso ( ), both of which are homes

[redacted]. Yet, Dr. Georgopoulos did not consider lead-based paint as a lead source when developing the hypothetical BLL framework and instead employed a single point estimate of 80 ppm for house dust lead levels.

The default dust lead value of 80 ppm assumed by class plaintiff's experts is also generally lower than the mean indoor dust lead concentrations measured in both urban and non-urban areas in the United States, irrespective of whether or not the homes contain lead-based paint. Frank et al. (2019) reviewed and compiled lead concentration data from various environmental media including indoor dust; they calculated a mean lead dust concentration of 176 ppm for floors and a mean dust concentration for all dust samples of 153 ppm. The default value of 80 ppm is also far lower than the mean dust lead levels measured in the homes of plaintiffs Kelso and Davis.

Obviously, the use of a default house dust lead level of 80 ppm artificially reduces the influence of house dust while artificially increasing the contribution of tap water to BLL in all age groups in the hypothetical BLL framework.

- *Incorrect assumptions regarding steady-state kinetics of BLLs under constant daily exposure*

In his deposition, plaintiff expert Dr. Georgopoulos made incorrect statements regarding the time required to reach a steady-state BLL under conditions of constant daily exposure. The time to reach a steady-state BLL is determined by the blood half-life, for which Dr. Georgopoulos states a value of "about a month" (Georgopoulos Depo 11/23/2022: p. 126, l. 2). After one half-life (approximately 30 days), 50% of steady-state is attained; after two half-lives (approximately 60 days), 75% of steady-state is attained; after three half-lives (approximately 90 days), 87.5% of steady state is attained (the EPA utilizes the duration of 90 days to define pseudo steady-state conditions in the IEUBK Pb Model); after four half-lives (120 days), 93.8% of steady-state is attained; and after five half-lives (150 days), 97% of steady-state is attained. As stated in the *Principals and Basic Concepts of Toxicokinetics*, "[i]n practice, a useful estimate of time to reach a steady-state is obtained by the following equation: time to 95% of steady-state = 4.3 x $t_{1/2}$ (days). For lead, the value would be 129 days (4.3 x 30)" (Gupta, 2016).

The relationship between actual and theoretical steady-state conditions is asymptotic (and thus over time, the difference in values become infinitesimally smaller). This is where Dr. Georgopoulos made two incorrect statements. First, Dr. Georgopoulos stated that "[i]f the exposure continues – because we're talking about the balance between what the organ is -- accumulates and what it excretes, then that will continue. Eventually we reach a plateau. But it will take much longer than a hundred days or ten days. I mean, it's going to be eventually -- it's a crisis. So eventually it will be substantially higher than what we calculate for 90 days" (Georgopoulos Depo 11/23/2022: p. 224, l. 5-13). That statement is incorrect. After 90 days, BLLs will have attained 87.5% of steady-state. From this point, BLLs cannot go "substantially higher" if the exposure remains constant. Had the plaintiff experts modeled an 8-month exposure period rather than 90 days, they would have found that the BLLs did NOT increase appreciably between the 3rd and 8th month. For example, the AALM-predicted BLL after 3-month of exposure to 10 and 100 ppb is already at 97% and 90% of the predicted BLL for 8-months of exposure, respectively. Second, when asked by counsel how long it takes to reach a plateau in BLL, Dr. Georgopoulos responded "[t]hat I don't know. It could take years" (Georgopoulos

Depo 11/23/2022: p. 224, l. 15). This statement is also incorrect. The general scientific consensus for time to steady-state is 4 to 5 half-lives, or, as previously noted above, is 4.3 half-lives, or 129 days, for lead.

Opinion 3: The hypothetical BLL framework developed by plaintiff experts contradicts the Flint adult BLLs measured during the switchover period and does not accurately characterize tap water lead exposures from February 10, 2015, to October 16, 2015.

The hypothetical BLL framework developed by plaintiff experts for Flint adolescents and adults is discussed in detail below. The BLL framework outputs for females as reported by plaintiff experts are shown below in Table 5.

Table 5. *Table 1 of Dr. Georgopoulos's expert report of BLL estimates (µg/dL) from the AALM-Leggett model: 90 days of exposure of female subjects 10-65 years old for representative WLLs (µg/L).*

	WLL (µg/L)	BLL (µg/dL) Age (years) - Female Subjects														
		10	12	14	16	18	20	25	30	35	40	45	50	55	60	65
Base	1.00	1.26	1.07	1.00	1.14	1.44	1.73	2.27	2.32	2.34	2.38	2.43	2.49	2.50	2.51	2.52
	2.00	1.28	1.08	1.01	0.15	1.46	1.75	2.31	2.36	2.38	2.43	2.48	2.53	2.55	2.56	2.56
	3.00	1.29	1.10	1.03	1.17	1.48	1.77	2.35	2.41	2.43	2.47	2.52	2.58	2.59	2.60	2.60
	5.00	1.33	1.12	1.05	1.19	1.51	1.82	2.42	2.49	2.51	2.56	2.62	2.68	2.69	2.68	2.67
	10.00	1.41	1.19	1.11	1.26	1.59	1.92	2.61	2.69	2.73	2.79	2.85	2.92	2.91	2.90	2.87
	20.00	1.58	1.32	1.23	1.39	1.76	2.14	2.99	3.11	3.17	3.25	3.33	3.40	3.37	3.32	3.25
	50.00	2.08	1.73	1.59	1.79	2.27	2.78	4.14	4.34	4.47	4.62	4.74	4.85	4.74	4.60	4.41
	100.00	2.92	2.40	2.19	2.45	3.11	3.86	6.05	6.40	6.65	6.90	7.10	7.26	7.02	6.72	6.34
	200.00	4.60	3.74	3.39	3.77	4.78	6.00	9.73	10.31	10.75	11.12	11.50	11.75	11.33	10.81	10.11
	300.00	6.28	5.08	4.60	5.09	6.46	8.15	12.97	13.75	14.37	14.94	15.41	15.79	15.23	14.52	13.59

As noted earlier, these results were generated via the AALM model by assuming default, "fixed" variables for all non-tap water exposure pathways; for tap water consumption the WLLs were varied as indicated in the above table. I will note there is at least one error in the table: the BLL of 0.15 µg/dL for 16 years of age at 2 ppb WLL (the correct BLL value is 1.15 µg/dL).

Plaintiff experts' hypothetical BLL framework purports to show that Flint residents experienced a sustained and "quantifiable" increase in BLLs as a function of increasing WLLs during the switchover period, even at very low WLLs. However, as described below, the methodology used is not valid conceptually and it ignores the actual weight of evidence regarding decreased tap consumption during the switchover period.

As initially mentioned in Opinion #1, by simply "fixing" all pathways (except the tap water pathway) to single point estimates, the plaintiff experts essentially accomplished nothing more than a one-

sided sensitivity analysis of the tap water contribution to total lead exposure. This masks the influence of the truly important variables while artificially magnifying the influence of WLL on BLL. In reality, and as described in this opinion, small increases in WLL will have no sustained "quantifiable" influence on a person's BLL because any such influence is mitigated by the much more significant and more variable influences from diet, soil, and house dust. I use the term "one-sided" sensitivity analysis here because plaintiff experts only considered assumptions that hypothetically *increased* the BLL relative to baseline (i.e., increasing WLLs) and ignored those that *decreased* the BLL relative to baseline (e.g., decreasing tap water consumption). Nonetheless, as described below, even the plaintiffs' biased (one-sided) analysis essentially proves that tap water consumption has little influence on BLLs at the WLLs that are relevant to Flint.

- *The hypothetical framework is proof that very few if any Flint residents had an elevated BLL during February 10, 2015, to October 16, 2015*

The hypothetical framework indicates that the BLLs of Flint females would not exceed 5 µg/dL unless the WLLs were greater than 50 to 200 ppb (>50 ppb for ages 10-18 and >200 ppb for ages 20 and older). As noted earlier, both of these WLLs are beyond the 95%ile of the distribution of tap WLLs in Flint. By definition, this means that the hypothetical framework does not apply to a vast majority of Flint residents. It also means that a vast majority of Flint residents did not have elevated BLLs according to the CDC definition of an "elevated BLL" for adults: 5 µg/dL (CDC, 2021).

- *The hypothetical BLL framework directly contradicts the known facts regarding adult Flint BLLs during the switchover period*

There are two publicly available analyses that evaluate whether measured adult Flint BLLs were statistically significantly increased during the switchover period (vs. pre-switchover). These are discussed below.

  - **Gómez, H. F., Borgialli, D. A., Sharman, M., Weber, A. T., Scolpino, A. J., Oleske, J. M., & Bogden, J. D. (2019). Blood Lead Levels in Females of Childbearing Age in Flint, Michigan, and the Water Crisis (2019). *Obstetrics and gynecology*, *134*(3), 628–635.**

Gomez et al. (2019) performed a retrospective cross-sectional analysis in which the authors assessed the geocoded BLLs of Flint females of childbearing age (ages 12-50 years old) during three matched 18-month timeframes. These timeframes included: Period I – April 25, 2012, to October 15, 2013 (before the switchover period), Period II – April 25, 2014, to October 15, 2015 (during the switchover period), and Period III – April 25, 2016, to October 15, 2017 (after the switchover period).

Gomez et al. (2019) reported that the mean Flint BLLs actually *decreased* during the switchover period (from 0.69 µg/dL to 0.65 µg/dL), albeit not to a degree that was statistically significant. Gomez et al. (2019) concluded that "[b]lood lead levels in Flint females of childbearing age did not increase during the Flint River water exposure and subsequent 18-month time period". In addition, the

authors stated that all of the individuals tested had BLLs that were well below the CDC reference level of 5 µg/dL.

The fact that mean BLLs did not increase during the switchover, in addition to the fact that the mean BLL reported in Gomez et al. (2019) (0.65 µg/dL) is well below all of the BLLs predicted by plaintiff experts for adult females during the switchover period (including even the pre-switchover baseline BLLs), directly contradicts the plaintiff experts' hypothetical BLL framework. The study by Gomez et al. (2019) was never mentioned or cited in any of the plaintiff expert reports, although clearly the plaintiff experts were aware of the study.

- **MDHHS (2016). 2014 Annual Report on Blood Lead Levels on Adults in Michigan. *Adult Blood Lead Epidemiology Surveillance (ABLES) Program, 1-62.***

A summary of data relating to % elevated blood lead levels (%EBLLs) in 2014-2015 in Flint adults was also presented by the Michigan Departmental of Health and Human Services (MDHHS, 2016). The authors performed an analysis of changes in the blood lead levels in adults (individuals ≥ 16 years old) living in the Flint zip codes 48501 through 48507. The following time periods were assessed: January-March 2014, April-December 2014, and January-September 30, 2015. The authors reported that there was no statistically significant increase in %EBLL (≥ 5 µg/dl) at any point during the switchover period, including the January-September 30, 2015 period that is relevant to this case.

I am not aware of any study that purports to have observed a statistically significant increase in either mean BLL or %EBLL in Flint adults during the switchover period. Both studies that evaluated adult BLLs for statistical significance during the switchover vs pre-switchover periods found no increase in BLLs during the switchover. These findings are consistent with the fact that tap water consumption significantly decreased during the switchover period. Hence, the weight of evidence clearly contradicts plaintiff expert claims that increased adult BLLs from tap water consumption during February 10, 2015, to October 16, 2015, increased the risk of lead-related health effects.

- *The hypothetical BLL framework is a worst-case scenario that is inconsistent with known facts and plaintiff expert testimony, and it applies to few if any adult Flint residents*

To construct the hypothetical BLL framework, plaintiff experts assumed 90 days of continuous tap water consumption at the generic, default consumption rates listed in the AALM model. This is inconsistent with the fact that, as even plaintiff experts acknowledged, tap water use by Flint residents was reduced by 70% by 2015. Hence, the hypothetical BLL framework is a "worst-case scenario" that only applies to individuals who continued to consume tap water daily at "normal" volumes (approximately one liter per day for adults) throughout February 10, 2015, to October 16, 2015. Many and perhaps most Flint adults (including the class representatives in this case) only used tap water for cooking and bathing from February 10, 2015, to October 16, 2015. Therefore, the "increased" BLLs in the framework do not apply to the class representatives, do not apply to a vast majority of Flint residents, and in fact may not apply to *any* Flint residents.

Also, given the facts of the case, the predicted BLL values in the framework are not representative of the class BLLs during February 10, 2015, to October 16, 2015. For example, it is implausible that a 20-year-old female with 3 ppb WLL in her tap water will have the predicted BLL of 1.77 µg/dL suggested by plaintiff experts, because that predicted BLL assumes full consumption of normal tap volumes (one liter per day is the equivalent to drinking a full quart of water from the tap) every day for 90 days. Instead, that 20-year-old female will have a BLL lower than 1.77 µg/dL because she only consumed tap water intermittently and/or at a reduced volume on those days when consumption actually occurred. Because the framework does not accurately represent the BLLs associated with different WLLs for Flint adults, it raises concerns as to whether and how plaintiff experts can rely on this table to reach any conclusions about potential "injuries" associated with tap water consumption. Specifically, if the BLLs are not representative or accurate, then it begs the question as to how any of the plaintiff experts can rely on this table for their causation conclusions regarding the class.

Finally, it is known that few Flint adults in 2015 had a BLL of > 5 µg/dL. Specifically, as summarized in Table 3 of a 2021 report by the MDDHS, only 15 of 203 Flint adults had a BLL of > 5 µg/dL (MDHHS, 2021). The vast majority of these were highly likely to be from occupational exposures (Rosenman and Stanbury, 2020). Hence, most of the BLL framework, i.e., any BLL greater than 5 µg/dL, does not apply a great majority of Flint adults.

Dr. Georgopoulos acknowledged in his deposition that he could not be certain that the hypothetical BLL framework actually applied to any particular individual in Flint. Based on the above, in my opinion it is certain that the framework does NOT apply to most or perhaps any individual in Flint. In other words, in addition to the fact that the framework doesn't apply to every single Flint resident, it likely doesn't apply to *any* Flint individuals.

- *A more realistic application of plaintiff's methodology shows that most Flint BLLs actually decreased during the February 10, 2015 to October 16, 2015 period*

As noted earlier and as testified to by Dr. Georgopoulos, tap water consumption rates significantly *decreased* during the switchover period and remained at a decreased level throughout the February 10, 2015 to October 16, 2015 period. This fact alone means the hypothetical BLL framework is largely irrelevant because in constructing the framework, the plaintiff experts assumed there was no decrease whatsoever in tap consumption rates during the switchover period. Had the plaintiff experts assumed a decreased tap consumption rate during the February 10, 2015 to October 16, 2015 period (e.g., the 70% reduction testified to by Dr. Georgopoulos), they would have found that BLLs did NOT increase during the switchover even at WLLs up to 3 ppb (Table 6). Given that most Flint individuals were exposed to less than 3 ppb WLL, the BLL framework is simply unworkable for describing a class that allegedly suffered injury from tap water exposure.

Table 6. *Comparison of BLLs with default and reduced water ingestion rates over a 90-day exposure period for 10-, 30-, and 50-year-old females.*

	Baseline		Reduced water ingestion rate (by 70% of default) during 90-day exposure at 3 ppb WLL	
Age	**WLL**	**BLL**	**WLL**	**BLL**
10	1	1.26	3	1.26
30	1	2.32	3	2.32
50	1	2.49	3	2.48

If it is further assumed that consumption is only intermittent (e.g., once per week), then (for example) for a 50-year-old female, the BLL would not be increased over baseline (2.49 µg/dL) even at WLLs up to 20 ppb.

These findings are actually consistent with Dr. Georgopoulos's acknowledgement that reduced consumption led to reduced adult BLLs during the switchover period. Specifically when asked by counsel about the decrease in adult BLLs reported in the Gomez et al. (2019) paper (which occurred during the switchover period), Dr. Georgopoulos stated "that th[e authors] include ten months where the consumption of tap water had been decreased by 70 percent. So…of course you would expect the blood levels to go down" (Georgopoulos Depo 11/23/2022: p. 178-179).

- *Failure to evaluate the variability and sensitivity of influential exposure pathways led to a false narrative that trivial increases in WLL exposures will result in sustained and "quantifiable" increases in BLLs*

Starting with the same AALM baseline assumptions used by plaintiff experts, a 10% increase in dietary lead exposure for a 30-year-old female results in a 5% increase in the BLL; however, the same increase (10%) in the WLL only results in a 0.4% increase in BLL (from 2.32 to 2.33 µg/dL).

Table 7. *Preliminary sensitivity analysis of lead exposure pathways assuming 10% increase in each individual exposure pathway*

Scenario	Dietary Lead Intake - Age 25 to 30 (µg/day)	Air (µg/m^3)	Dust (ppm)	Soil (ppm)	WLL (µg/L)	BLL (µg/dL)	Percent Increase in BLL
Baseline	13	0.1	80	100	1	2.32	--
+10% Food Lead Intake	14.3	0.1	80	100	1	2.44	5.0%
+10% Increase in Air Lead Conc.	13	0.11	80	100	1	2.39	2.9%
+10% Dust Lead Conc.	13	0.1	88	100	1	2.33	0.4%
+10% Soil Lead Conc.	13	0.1	80	110	1	2.33	0.4%
+10% Water Lead Level	13	0.1	80	100	1.1	2.33	0.4%

As can be seen in Table 7 above, tap consumption is in fact one of the *least* influential of all adult lead exposure pathways. Yet tap consumption is the only pathway for which plaintiffs considered multiple different values (the WLLs). All of the truly influential variables (e.g., dietary lead ingestion) were treated as fixed, single point estimates within each age group. This masks the influence of the truly influential variables while at the same time artificially magnifying the relatively trivial influence of WLL. In reality, increases in WLL will have relatively little influence on a person's dose (BLL) because any such influence is mitigated by the much more significant and more variable influences from the other pathways. In fact, even large changes in WLL will have little influence on BLL relative to much smaller changes in other, more influential pathways.

For example, starting with the same AALM baseline assumptions used by plaintiff experts, a 200% increase in the WLL (from 1 ppb to 3 ppb) results in a BLL increase of 6.7% (from 2.32 to 2.48 µg/dL) (Table 8). Yet a much smaller (30%) increase in the dietary lead exposure (from 13 ug/day to 16.9 ug/day) results in an 18% BLL increase (from 2.32 to 2.74 µg/dL). I specifically chose the hypothetical WLL of 3 ppb because as described earlier, in 2015 most Flint residents were exposed to WLLs of 3 ppb or less. The assumed 30% increase in dietary lead exposure is well within the normal range of adult dietary lead intake.

Table 8. *Percent changes in BLL for 30-year-old females due to changes in assumed WLL or dietary lead exposures*

Scenario	Dietary Lead Intake – Age 25 to 30 (µg/day)	WLL (ppb)	Air (µg/m^3)	Dust (ppm)	Soil (ppm)	BLL (µg/dL)	% Change in BLL
Baseline diet + 1 ppb WLL	13	1	0.1	80	100	2.32	--
Baseline diet + 3 ppb WLL	13	3	0.1	80	100	2.48	6.7%
30% Decrease in diet - 9.1 µg/day	9.1	1	0.1	80	100	1.91	-18%
30% Decrease in diet - 9.1 µg/day	9.1	3	0.1	80	100	2.07	-11%
30% Increase in diet - 16.9 µg/day	16.9	1	0.1	80	100	2.74	18%

It is also critical to note that the trivial BLL increases associated with large WLL increases are completely cancelled out by modest decreases in dietary lead consumption. Specifically, as can be seen in Table 7, a 200% WLL increase (3 ppb) in conjunction with a 30% decrease in dietary lead consumption (9.1 ug/day) results in an 11% *decrease* in BLL.

Even the exposure pathways that involve daily ingestion of a very small amount of material, such as soil and house dust ingestion, influence total lead exposure and associated BLLs more than tap water ingestion. This is because WLL is present in parts per billion (ppb) levels, while lead levels in soil and dust are present in parts per million (ppm) levels. Hence, although an adult may ingest far more tap water on a daily basis than they do soil or dust, the soil and dust contain lead concentrations that are thousands of times higher than the lead levels in the tap water. For example, Figure 9 below depicts the mean lead levels measured in the dusts and soils at the home of class plaintiffs Davis versus the median of the August 2015 first draw samples from copper service lines (the Davis household had copper service lines).

Figure 9. *Comparison of Mean Lead Levels in House Dust and Soil of Davis residence vs Median 2015 Flint Tap Water from Copper Service Lines*

It is interesting to note that the Davis residence, as well as the residence of the Kelso plaintiffs, were recently [redacted]. None of the expert reports from plaintiff experts mention this fact.

Given the relatively greater influence diet and soil/dust sources have on a person's BLL (relative to tap water consumption), a small increase in tap WLL is simply not going to result in a "quantifiable" increase in average BLLs in a population of Flint adults. This is consistent with the aforementioned fact that Gomez et al. (2019) reported the mean BLL in adult Flint women during the switchover was not increased.

As noted earlier, the alleged "increased" BLLs in plaintiff's hypothetical BLL framework are simply an artifact of treating a single exposure pathway as a variable while ignoring the variability in all the other exposure pathways. Plaintiff's methodology more accurately represents a sensitivity analysis, which is merely a tool used to evaluate the degree to which a model output changes in response to changes in specific input assumptions. The output of a sensitivity analysis is not intended to be used to assess an individual's true exposure via all pathways combined nor is it intended to be used to reach health risk conclusions about that individual. I am not aware of any regulatory health risk assessment guidance document that suggests a sensitivity analysis of a single exposure pathway can be substituted for an actual quantitative health risk assessment (nor do plaintiff experts cite to any such document).

- *Despite plaintiff experts' claims, a majority of the predicted BLL increases in their framework are in fact not "quantifiable"*

Plaintiff experts claim that all the increased BLLs in Table 5 are "quantifiable". I have summarized the actual BLL increases in Table 9 below.

Table 9. *Summary of increases in female BLLs over baseline by age using predicted BLLs reported by plaintiff experts*

Increases in BLL estimates compared to the baseline BLL estimates (Predicted BLL estimates (µg/dL) - Baseline BLL estimates) from AALM-Leggett model for 90 days exposure of female subjects 10-65 years old for representative WLLs (µg/L)																
		BLL (µg/dL)														
	WLL	Age (years) - Female Subjects														
	(µg/L)	10	12	14	16	18	20	25	30	35	40	45	50	55	60	65
Base	1.00	1.26	1.07	1.00	1.14	1.44	1.73	2.27	2.32	2.34	2.38	2.43	2.49	2.50	2.51	2.52
	2.00	0.02	0.01	0.01	0.01	0.02	0.02	0.04	0.04	0.04	0.05	0.05	0.04	0.05	0.05	0.04
	3.00	0.03	0.03	0.03	0.03	0.04	0.04	0.08	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.08
	5.00	0.07	0.05	0.05	0.05	0.07	0.09	0.15	0.17	0.17	0.18	0.19	0.19	0.19	0.17	0.15
	10.00	0.15	0.12	0.11	0.12	0.15	0.19	0.34	0.37	0.39	0.41	0.42	0.43	0.41	0.39	0.35
	20.00	0.32	0.25	0.23	0.25	0.32	0.41	0.72	0.79	0.83	0.87	0.90	0.91	0.87	0.81	0.73
	50.00	0.82	0.66	0.59	0.65	0.83	1.05	1.87	2.02	2.13	2.24	2.31	2.36	2.24	2.09	1.89
	100.00	1.66	1.33	1.19	1.31	1.67	2.13	3.78	4.08	4.31	4.52	4.67	4.77	4.52	4.21	3.82
	200.00	3.34	2.67	2.39	2.63	3.34	4.27	7.46	7.99	8.41	8.74	9.07	9.26	8.83	8.30	7.59
	300.00	5.02	4.01	3.60	3.95	5.02	6.42	10.70	11.43	12.03	12.56	12.98	13.30	12.73	12.01	11.07

The term “quantifiable”, although never defined by any of the plaintiff experts, means “can be measured”. Yet as can be seen in Table 9 a majority of the “increased” BLL values are so trivial they are certainly not quantifiable according to the proper definition of the term.

For example, below a WLL of 3 ppb, the BLL increases over baseline were less than 0.1 µg/dL. Such negligible increases are well within the daily variation in BLL that occurs in all individuals and more importantly they cannot be clinically detected because of the analytical variability associated with the methods used to measure BLL. The current Clinical Laboratory Improvement Amendments (CLIA) acceptability criteria for accuracy in BLL measurements is ±4 µg/dl for BLLs <40 µg/dl and the best reported accuracy for BLL measurements is ±2 µg/dL (Caldwell et al., 2017). Using the CLIA acceptability criteria of ±4 µg/dl, a BLL measurement cannot detect a difference between a female exposed to baseline WLL vs a female exposed to WLLs as high as 100 ppb.

In addition, assuming a standard deviation of 1.8 for each of the BLLs summarized in Table 8 (this is the standard deviation as defined in the EPA’s “Adult Lead Methodology” biokinetic model), for all age groups the WLL must be greater than 20 ppb to yield an estimated BLL that is actually statistically significantly greater than the baseline BLL.

It is worth noting that Table 5, taken from the report of Dr. Georgopoulos, and Table 9 confirm that tap WLL consumption contributes very little to total BLL at all WLLs that could be considered relevant to the vast majority (>95%) of the Flint population.

Finally, as discussed earlier tap water ingestion contributes only a fraction to the total BLL, which Dr. Hu acknowledged: “the declaration pertains to any individual who has been exposed to lead, assuming that one could then try to **determine what the estimated contribution of the corrosive water was to the blood lead levels”** (Hu 11/21/2022: p. 41). As can be seen in Table 9, tap WLLs of up to 50 ppb do not contribute more than 5 µg/dL to the total BLL and therefore tap ingestion alone does not create an elevated BLL (per the CDC definition of “elevated”) at any WLL that is relevant to the vast majority of the Flint residents. Further, in order to properly answer the question raised in Issue #3, one would have to evaluate the BLL associated with *tap consumption alone*, not BLLs that are mostly driven by soil, diet, and house dust.

In summary, since many of the plaintiff experts' estimated doses (BLLs) for the class are not actually increased over baseline in any quantifiable manner, then it follows that, for a significant portion of the BLL framework, one cannot reach a dose-related finding of "causation" for any of the alleged health effects.

- *The hypothetical BLL framework does not apply to the February 10, 2015 to October 16, 2015 period*

There are several reasons why the hypothetical BLL framework does not apply to the relevant time period. First and foremost, as discussed by plaintiff experts and in published studies of Flint residents, as well as in testimony from named class plaintiffs, Flint tap water consumption significantly decreased by 2015. This is not surprising, given the disagreeable odor, color and taste of the water. Yet plaintiff experts did not consider reduced consumption rates when developing the BLL framework and therefore the BLLs presented in the framework are inaccurate estimates of the February 10, 2015 to October 16, 2015 Flint adult BLLs associated with any particular WLL. For example, as noted earlier, the BLLs at up to 3 ppb WLL during the switchover were actually at or *below* baseline BLLs, not above, when the tap consumption rate is reduced by 70%.

Second, the BLL framework is based on the invalid assumption that Flint tap water had non-detectable WLLs before the switchover. This invalid assumption was used to create the baseline BLL values against which all the other estimated BLLs were compared. Because the baseline BLLs are not correct, the entire BLL framework (and any estimate of lead dose/duration based on that framework) is invalid. Third, the BLL framework only considers *increased* WLLs during the switchover; the evidence actually indicates that the Flint tap WLLs were *lower* during February 10, 2015 to October 16, 2015 period than pre-switchover. Increased WLLs might apply to 2014 but they do not apply to any period of 2015. Finally, some individuals installed filters in 2015 that were not present in 2014. Indeed, class representative Ms. Rhonda Kelso testified [redacted].

- *Only a small fraction of the hypothetical BLL framework would actually apply to Flint residents because WLLs at most residences were very low or non-detectable*

As part of an effort to mitigate the switchover effects of increased lead release from the lead and galvanized service lines, the City of Flint began the FAST Start initiative in March 2016. The FAST Start initiative is a program designed to identify and replace public and private service lines that are made of lead or galvanized steel around the city. At the beginning of the FAST Start program, a statistical model combined with active learning methods was used to optimize the identification of homes with lead or galvanized public or private service lines that needed to be replaced (Abernethy et al., 2018). The initial statistical model used existing water lead monitoring data, date of home construction, home value, and existing service line information (Abernethy et al., 2018). As the program proceeded, the model was updated using an active learning algorithm to further optimize the results based on the homes already explored as part of the program. Use of this method means that the sample of homes identified in the program for service line exploration are biased towards homes with lead or galvanized service lines and not those with copper service lines.

As of November 24, 2021, 25,948 homes have participated in the FAST Start program. A total of 16,376 (63.1%) of the homes did not have any lines replaced, and this is primarily due to the fact that well over half (15,969, or 61.5%) of the homes had copper public and private lines. A little over 9,500 homes (9,571, or 36.9%) had the public or private service line or both service lines replaced because either one or both were lead or galvanized pipe. Because the FAST Start program prioritized homes with lead-containing service lines for exploration, the percent of homes with lead or galvanized public or private service lines that needed to be replaced for all 55,000 homes within the City of Flint is expected to be actually lower than 36.9%, i.e. it is highly likely that most of the 30,000 homes not yet inspected do not have lead-containing service lines as depicted in Figure 10.

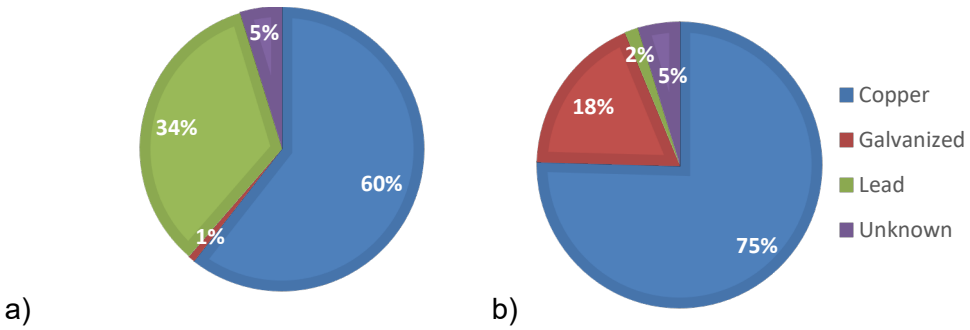


Figure 10. *Composition of a) public service lines and b) private service lines as identified by the FAST Start program.*

As noted earlier, tap WLLs measured in copper service lines during the February 10, 2015 to October 16, 2015 period were more likely than not to be non-detectable. Accordingly, a Flint adult was more likely than not to have non-detectable WLLs at their residence during that time period. Hence, by defining a class as "[a]ll persons...who, for any period of time between February 10, 2015, and October 16, 2015 were exposed to…drinking water supplied by the City of Flint," for a large fraction of the Flint adult population, plaintiff experts have assumed lead exposure via tap consumption when in fact the evidence indicates no such exposure occurred.

- *The weight of evidence indicates that class representatives and most Flint residents did not have elevated BLLs during February 10, 2015 to October 16, 2015*

[redacted]

[redacted]

Table 10. *Summary of AALM input parameters and predicted BLL outputs.*

	Parameter	Unit	Barbara Davis	Darrell Davis
Environmental Input Parameters	Composite Switchover WLL	µg/L	[redacted]	[redacted]
	Soil lead (excluding dripline)	ppm	[redacted]	[redacted]
	Dust lead (excluding attic)	ppm	[redacted]	[redacted]
	Baseline WLL	µg/L	[redacted]	[redacted]
	Diet	µg/day	[redacted]	[redacted]
	Air	µg/m3	[redacted]	[redacted]
Biological Input Parameters	Age	years	[redacted]	[redacted]
Output Parameter	Predicted BLL	µg/dL	[redacted]	[redacted]

I calculated the percent contribution of each environmental lead source to BLL for Barbara and Darrell Davis. As the individuals were the same age and had the same exposure concentrations, the source contribution profiles were similar for both plaintiffs as summarized in Figure 11 below. It can be observed from this figure that diet, indoor dust, and air are the primary drivers of BLL and that tap water consumption has very little influence on BLL.

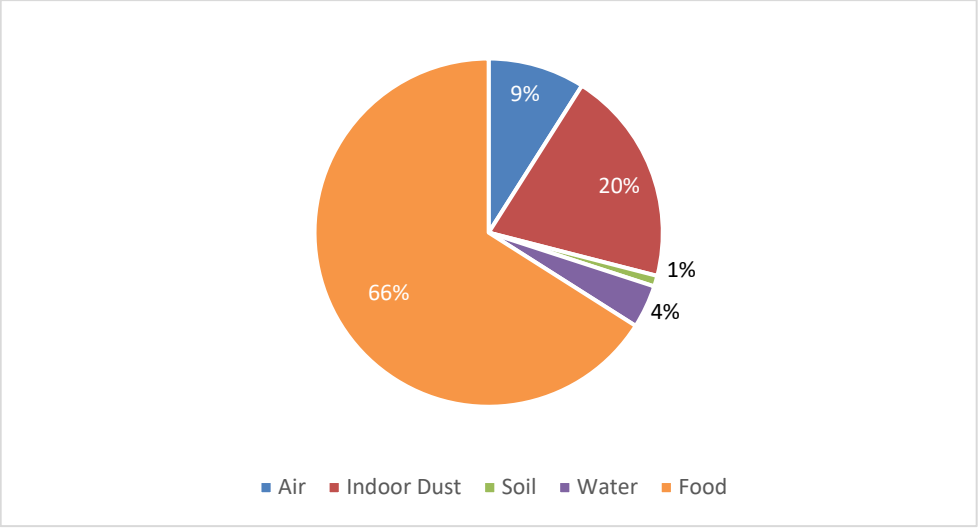


Figure 11. *Percent source contributions of diet, dust, soil, water, and air to the BLLs of Barbara and Darrell Davis.*

It is clear that in February 10, 2015 to October 16, 2015 the class representatives did not have elevated BLLs nor is there any evidence that their BLLs were increased “quantifiably” or in any other manner during that time. The same is true for all other BLLs I have reviewed for other adults named

in the documents prepared by plaintiff's counsel. I use the term "elevated" here in direct reference to the CDC reference level of 5 µg/dL for adults; adult BLLs below 5 µg/dL are not considered to be elevated by the CDC (CDC, 2021). The term also applies to the baseline BLL of each adult before the switchover: there is no evidence to indicate that the BLLs of the class representatives in February 10, 2015 to October 16, 2015 were greater than during the pre-switchover period.

This is also true for a majority and perhaps all of Flint residents during February 10, 2015, to October 16, 2015. As noted earlier, the mean measured BLL in Flint adult women during the switchover was 0.65 µg/dL (Gomez et al., 2019). This is far below the CDC reference level of 5 µg/dL for adults. Similarly, Roy and Edwards (2021) recently published estimated mean BLLs for adult Flint women before, during, and after the switchover period. Even though the authors employed very high tap WLL estimates (90th %ile) and full tap water consumption rates, the mean BLLs were less than 2.5 µg/dL during February 10, 2015, to October 16, 2015.

Roy and Edwards (2021) recently used the International Commission on Radiological Protection (ICRP) Leggett model to conduct that work; this model was influential in setting the computational framework in the current Version 2 of AALM (EPA, 2019; Pounds and Leggett, 1998). A current version of the ICRP model (v R5.CHELATE.3000) was obtained from Syracuse Research Corporation by Roy and Edwards (2021) to estimate blood lead concentrations of a typical woman consuming Flint tap water from 2011 through 2017. They used estimates of the 90th percentile tap water WLL and the community consumer-only water intake rate currently recommended by the U.S. EPA (2019) of 1.2 L/day. As shown in Figure 12 and Table 11, the current ICRP and AALM models predict similar BLLs when the Roy and Edwards WLL and water intake rates are entered into the AALM modeling spreadsheet used by the plaintiff experts. Similarly, the Roy and Edwards (2021) ICRP lead intake to BLL ratio of approximately 0.08 (µg/dL)/(µg/L) for the model shown is similar to the ratio in the plaintiff experts' hypothetical BLL framework after 90 days of exposure of 0.05 and 0.06 (µg/dL)/(µg/L) for a 20- and 25-year old female, respectively. Thus, for similar exposure factors, the AALM model used in the plaintiff experts' hypothetical BLL framework predictions is expected to produce similar predictions as the current ICRP model used in the Roy and Edwards (2021) study. Both models predict that BLLs would not be elevated during the switchover period.

Table 11. *Comparison of Roy and Edwards (2021) ICRP model predictions to a) AALM model predictions for same exposure factors including 90th percentile WLL and water intake and (b) plaintiff experts' estimated 90-day exposure of female subjects to hypothetical WLL.*

Year	Female Age	WLL (µg/L)*	Average Pb Intake (µg/day)**	ICRP Average BLL (µg/dL)*	AALM BLL (µg/dL)
a) Flint 90th Percentile WLL and Average BLL					
2011	20	35	43	3.3	2.5
2012	21	20	24	2.3	2.0
2013	22	23	27	2.4	2.5
2014	23	27	33	2.6	3.2
2015	24	17	20	1.8	2.6
2016	25	16	19	1.5	2.4
2017	26	13	16	1.4	2.4
2011-2017	20-26	22	26	2.2	2.5
b) Hypothetical WLL and BLL after 90 Days of Exposure					
90 days exposure of female subjects to hypothetical WLL [Table 1 of Dr. Georgopoulos's expert report]	20	10	37	--	1.9
		20	43	--	2.1
		50	61	--	2.8
	25	10	40	--	2.6
		20	47	--	3.0
		50	67	--	4.1

*Table S4 in Roy and Edwards (2021) provided intake in µg/day; the intake was used with 1.2 L/day to calculate WLL in µg/L
**90th percentile WLL scenario tap water only; hypothetical scenario includes background intake from air, dust, soil, food in addition to tap water.

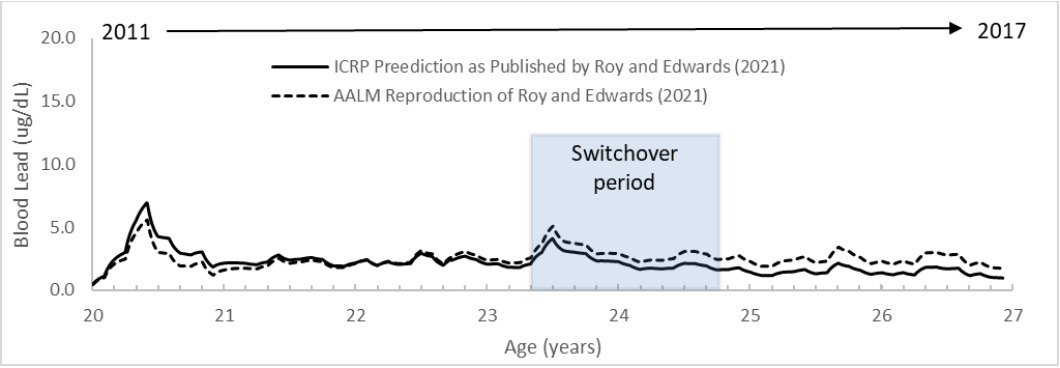


Figure 12. *Comparison of Roy and Edwards (2021) ICRP central tendency model predictions shown in authors' Figure 2 and presented in Table S4 vs AALM predictions for same Flint 90th percentile WLL, daily water intake, and period.*

- *The hypothetical BLL framework cannot be applied to any particular Flint individual*

On pages 22-23 of Dr. Georgopoulos's report, regarding the BLLs in his hypothetical framework, he notes that they "can provide a means for classifying categories of exposure that pertain to different levels of adverse impacts on health…if subject-specific information is available then it could be used in the calculations for case-specific estimates...".

The hypothetical BLLs developed by Dr. Georgopoulos cannot be applied to any particular Flint individual because the requisite "subject site information" mentioned by Dr. Georgopoulos (e.g., dust and soil lead concentrations and WLLs) simply does not exist for any Flint individual. Dr. Georgopoulos acknowledged in his deposition that he was not sure if the BLL framework applied to any single individual in Flint. This is a critical flaw in plaintiff experts' methodology because many of the "increased BLLs" in their framework are not actually true increases and/or they are far too low to represent a potential injury. Since there is no possible valid methodology to accurately place any particular individual into the framework, in addition to the fact that framework only evaluated a "worst-case scenario" that does not apply to most or perhaps any individual in Flint, the framework serves no real purpose.

Opinion 4: Dr. Hu's claims of lead-related health effects that allegedly could have occurred due to tap water exposure from February 10, 2015 to October 16, 2015 are not supported by the scientific evidence.

Introduction

Dr. Hu specifically addressed the question defined by the court as "Issue 3: Were the contaminated water conditions capable of causing harm to Flint residents, properties, property and businesses?". Dr. Hu concluded that there was sufficient evidence to answer this question in the affirmative and he cited to several papers in his analysis to address the following health endpoints: cardiovascular, neurological, renal, hematological, immunological, and reproductive.

As discussed earlier, understanding exposure duration is key to understanding the potential risk of adverse health effects in any exposure setting. It is a well-established principle of toxicology and epidemiology that adverse health effects that occur due to chronic chemical exposures (several years) cannot be assumed to occur following acute (days) or sub-chronic (e.g., less than a year) exposures. Essentially, the lower the exposure, the longer it takes to reach the minimum threshold dose required for causation. This is why, for example, the EPA sets different acceptable doses for chronic and sub-chronic chemical exposures; the acceptable dose for the chronic exposure is always more stringent than for the corresponding sub-chronic exposure.

Therefore, to answer Issue #3 one must consider both dose (BLL) and duration (the length of the exposure period) for each alleged health endpoint. Dr, Hu agreed that both dose and duration must be considered; in his declaration he stated that "[i]t is important to acknowledge that **depending on the magnitude and duration of exposure**, lead is capable of causing a wide range of impacts" (Hu Declaration: p. 8). Regarding exposure duration, a single measured adult BLL value by itself is not sufficient to understand one's total lead exposure because it is simply a snapshot that indicates whether the individual is being exposed or was recently exposed to elevated levels of lead. Instead, one needs to evaluate the length of time over which the dose (elevated BLL) occurred. As noted by the ATSDR, lead-related health effects that occur following extended environmental exposure periods do not necessarily occur over shorter exposure periods: "health effects of Pb in humans are considered to be associated with chronic exposure, rather than to shorter exposures (ATSDR, 2020: p. 431).

Dr. Hu agreed that a minimum lead dose (reflected by BLL) must be maintained over a minimum amount of time (exposure duration) to support a causation conclusion:

> In his deposition, when asked**, "[a]nd it may be that exposure at certain levels for 30 days, to pick a number, is enough but it - - it also may be that that's insufficient time, fair to say?," Dr. Hu responded, "I think that's fair"** (Hu 11/21/2022: p. 42, l. 22-25 – 43, l. 2). In addition, when asked whether it was **"fair to say that in determining whether or not lead could have caused any particular injury to a particular individual, first one has to determine the length of exposure and the amount of exposure, fair?," Dr. Hu responded, "Yes"** (Hu 11/21/2022: p. 56, l. 22 – p. 57, l. 3). Further**, when asked, "lead may cause some injuries but not others depending upon the - - the amount of lead involved and the length of exposure, fair to say?," Dr. Hu also replied, "Yes"** (Hu 11/21/2022: p. 56, l. 6-9).

Hence, for any of the alleged health effects in this case, to demonstrate that Flint tap water was "capable of causing harm to Flint residents" during the period of February 10, 2015 to October 16, 2015, it must be shown that the lead dose (BLL) during that 8 month time frame exceeded the minimum dose/duration causation threshold for the health effect under consideration. Dr. Hu acknowledged that he did not determine the minimum lead dose/duration threshold for any of the alleged health effects, opining that it is: "…**difficult to just say what a threshold might be**" (Hu 11/21/2022: p. 73, l. 15-24, p. 74, l. 1-5).

As described below, the doses (BLLs) and exposure durations in the studies cited by Dr. Hu are not relevant to the Flint residential population during February 10, 2015 to October 16, 2015. Further, the evidence indicates that lead dose/durations experienced by Flint residents were far too low to exceed minimum causation thresholds for any of the alleged health effects.

Dr. Hu stated in his declaration that he would be "focusing his question on the potential harm to Flint residents associated with exposure to the toxic metal lead as a result of ingesting Flint water for some duration between May 1, 2014 and October 16, 2015" (Hu Declaration: p. 2). However, the class in this case pertains to the time period between February 10, 2015 and October 16, 2015. It is unclear whether Dr. Hu was aware that the relevant time frame in this case is only 8 months, not 18 months.

Dr. Hu's views on the 90-day modeling period

Dr. Hu testified that the BLL modeling results provided to him by Dr. Georgopolous were for a 90-day period. He explained that if the exposure period were different, he would expect changes in the estimated BLLs. If Dr. Hu was suggesting that a longer modeling period would result in substantially higher BLLs, he is incorrect. As discussed earlier, after 90 days the BLLs will have attained close to 90% of steady-state conditions. If Dr. Hu was suggesting that a shorter exposure period would have resulted in lower BLLs, then he is correct. However as discussed in detail earlier, plaintiff experts made no effort to evaluate any other scenario beyond the worst-case scenario of continuous daily consumption at full default consumption rates for 90 straight days.

The studies cited by Dr. Hu involve BLLs that are far higher than any of the BLLs measured in Flint adults

In his declaration, Dr. Hu stated that the scientific literature "supports the view that relatively modest elevations in blood lead levels (i.e., below 5 ug/dL) can result in adverse effects on neurological, renal, hematological, immunological, and reproductive functions" (Hu Declaration: p. 19). Dr. Hu also cited to literature in support of a statement that "relatively modest elevations in blood lead levels – i.e., within 5 µg/dL…are a cause of clinically significant elevations in blood pressure as well as the risk of clinical hypertension, which, in turn, pose elevated risks of adverse cardiovascular outcomes (e.g., myocardial infarction, stroke)" (Hu Declaration: p. 8).

Dr. Hu infers that it is therefore reasonable to expect that these adverse effects could have occurred or will occur as a result of increased WLLs and increased BLLs during the 8-month time frame defined by the court. Of course, this assertion is directly contradicted by the empirical data that were available to Dr. Hu when he prepared his declaration: tap WLLs and adult mean BLLs were not increased relative to baseline pre-switchover conditions in the February 10, 2015 to October 16, 2015 time frame (neither of these findings were mentioned or even cited in Dr. Hu's declaration).

As previously discussed in Opinion #3, Gomez et al. (2019) reported a measured female adult mean Flint BLL of 0.65 µg/dL during the switchover period. This mean BLL value is even lower than the pre-switchover baseline BLLs estimated by Dr. Georgopoulos for Flint female adults (BLL range of 1.73-2.52 ug/dl). The measured mean BLL value of 0.65 µg/dl is also consistent with the fact that all BLLs from class representatives during February 10, 2015 to October 16, 2015 were non-detectable.

My review of the studies cited by Dr. Hu finds that, for all of the alleged health effect endpoints, central tendency BLLs measured in the cited cohorts were consistently higher than the measured mean of 0.65 µg/dl reported by Gomez et al. (2019), and the upper bound BLLs measured in the cohorts were consistently orders of magnitude higher than 0.65 µg/dl. For example, in a study evaluating the association between BLLs and **neurological** effects (cognitive functioning) in older U.S. adults, Przybyla et al. (2017) reported a geometric mean of 2.17 µg/dL and an upper bound of 16.4 µg/dL for the cohort. Specific to **renal** effects, Kim et al. (1996) reported a mean of 9.9 µg/dL and a maximum BLL of 54.1 µg/dL, and Harari et al. (2018) reported a median of 2.5 µg/dL and an upper bound BLL of 25.8 µg/dL. Dr. Hu cited to Sirivarasai et al. (2013) and Pizent et al. (2008), as evidence that elevations in BLLs are associated with **immunological** effects. Sirivarasai et al. (2013) reported a mean of BLL of 5.45 µg/dL and a maximum BLL of 24.63 µg/dL. Pizent et al. (2008) noted median values of 2.16 µg/dL and 3.17 µg/dL for women and men respectively, and corresponding upper bound BLLs of 7.35 µg/dL and 7.23 µg/dL. Specific to **reproductive** effects, Hu et al. (2006) reported mean BLLs in mothers (measured during first, second, and third trimesters and delivery) ranging from 6.08 to 7.26 µg/dL and corresponding upper bound BLLs ranging from 22.44 to 43.59 µg/dL. In this same study, mean BLLs for children (infant pairs, measured in cord blood and at 12 and 24 months) were reported to range from 4.79 to 6.20 µg/dL, with corresponding upper bound BLLs ranging from 20.0 to 36.8 µg/dL. Additionally, in terms of **cardiovascular** effects, Dr. Hu cited multiple studies that evaluated associations between biomarkers of lead exposure and blood pressure and/or hypertension, such as Gambelunghe et al. (2016) (mean BLL: 2.8 µg/dL;

maximum BLL: 25.8 µg/dL) and Yan et al. (2022) (geometric mean: 4.73 µg/dL; maximum: 58.2 µg/dL). Although Dr. Hu did not cite to any specific studies regarding **hematological** effects in his declaration, ATSDR (2020) reported data for multiple epidemiological studies of heme metabolism, blood hemoglobin/erythrocyte count, and other hematological effects at mean blood lead concentrations ≤ 10 µg/dL; of these studies, mean BLLs of up to 9.96 µg/dL were reported, in addition to maximum BLLs of up to 37.78 µg/dL (Ahamed et al., 2006; Liu et al., 2015).

None of the cited studies cited by Dr. Hu reported health effects at BLLs commensurate with those that existed in Flint residents in February 10, 2015 to October 16, 2015. There is no evidence in the cited papers to indicate that any of the alleged health effects occurred in a population with a mean BLL as low as 0.65 µg/dl, regardless of the exposure duration. Dr. Hu essentially agrees with this conclusion. When asked to confirm that he did not know of any individuals in Flint who actually have BLLs "that may be a cause of concern", Dr. Hu replied, "Correct" (Hu 11/21/2022: p. 42, l. 14-17). Additionally, Dr. Hu affirmed that some Flint individuals may not have experienced an increase in their WLLs at all (Hu 11/21/2022: p. 74), and he also acknowledged that an individual could have been living in Flint but not drinking the tap water (Hu 11/21/2022: p. 72).

Finally, as discussed earlier, tap water ingestion contributes only a fraction to the total BLL, which Dr. Hu acknowledged: "the declaration pertains to any individual who has been exposed to lead, assuming that one could then try to determine what **the estimated contribution of the corrosive water was to the blood lead levels**" (Hu 11/21/2022: p. 41). As described in Opinion #3, tap WLLs of up to 50 ppb do not contribute more than 5 ug/dl to the total BLL and therefore tap ingestion alone does not create an elevated BLL (per the CDC definition of "elevated") at any WLL that is relevant to the vast majority of the Flint residents. Further, in order to properly answer the question raised in Issue #3, one would have to evaluate the BLL associated with *tap consumption alone*, not BLLs that are mostly driven by soil, diet, and house dust. Dr. Hu did not do this; he instead evaluated the total estimated BLLs in Dr. Georgopoulos's hypothetical BLL framework, yet only a fraction of those BLL values are due to tap consumption even at WLLs well above baseline.

Dr. Hu relied on studies that do not have exposure durations relevant to the multi-defendant class

As noted above, Dr. Hu agreed that both dose and exposure duration are important, i.e., he acknowledged that the lower the BLL, the longer the exposure period would need to be to reach a minimum causation threshold. He also agreed that the exposure durations of studies he cited were chronic and occurred over several years

> When asked about the exposure durations in the studies he relied on relating to cardiovascular effects in adults and children reported in ATSDR 2020, Dr. Hu stated "**These studies all had exposure metrics that – that signify that <u>the exposure was probably years</u>**" (p. 134, l. 15-24)

I have evaluated the cited studies and Dr. Hu is correct. For each alleged health effect, the cited studies are primarily evaluations of cohorts exposed to elevated lead levels in the environment (soils and air), often for 10 years or more. Not one of these studies involved an exposure period anywhere near as brief as the 8-month period defined for the Flint class. As mentioned above, it is possible

that Dr. Hu was not aware of the specific 8-month exposure duration specified for the class because the time frame of February 10, 2015 to October 16, 2015 is not mentioned in his declaration.

Dr. Hu's analysis does not address the question posed in Issue #3 and does not support a general causation conclusion for any of the alleged health effects

Dr. Hu agrees that a minimum lead dose over a minimum period of time is required to cause each of the alleged health effects in this case. However, no evidence is offered by Dr. Hu or any other plaintiff expert to indicate whether the threshold lead dose/durations for any alleged health effect were exceeded by Flint residents. They specifically failed to demonstrate that any estimated or measured Flint adult BLL (dose), maintained over an 8-month period (duration), could have resulted in any of the adverse effects discussed by Dr. Hu. In the studies cited by Dr. Hu, the BLLs were far higher and the exposure durations far longer than any Flint resident would have experienced in Feb 10, 2015 to October 16, 2015. None of the cited studies (individually or in aggregate) are evidence of potential risk of adverse health effects in Flint residents. In short, plaintiff experts did not answer the question posed by Issue #3, i.e., they did not present an analysis that proves the Flint tap water was capable of causing harm to any Flint resident. Accordingly, for the time period February 10, 2015 to October 16, 2015 plaintiff experts cannot reach a dose-related finding of "causation" from tap water ingestion for any of the alleged health effects.

The AALM model predicts that bone lead levels in Flint adults would not have increased significantly even after 8-months of daily consumption of elevated WLLs

In his declaration in this case, Dr. Hu relied entirely on estimated BLLs as metrics of Flint tap water exposure. However, in his published papers, Dr. Hu has long advocated for the use of bone lead levels as a superior metric (relative to BLLs) for assessing cumulative lead exposure and general causation in adults. Therefore, I used the AALM model employed by plaintiff experts in this case to evaluate estimated bone lead levels in Flint residents. Specifically, I determined whether 8 months of tap water exposure would actually result in an increased bone lead level in a Flint adult. For the purpose of this exercise, I modeled the bone lead level of a 60-year-old female after 8 months of exposure to the baseline WLL of 1 ppb vs the elevated WLLs of 10 and 100 ppb. Model default values were used for all other exposure pathways (diet, etc.). The results are shown in Table 12 below.

Table 12. Cortical bone lead concentrations (ug/g) for a 60-year-old female with 8 months of exposure to baseline or elevated WLLs

Time	1 ppb WLL	10 ppb WLL	100 ppb WLL
Start of 8-month "pulse"	1.32	1.32	1.32
1 month after pulse start	1.32	1.32	1.33
2 months after pulse start	1.32	1.32	1.34
3 months after pulse start	1.32	1.32	1.36
4 months after pulse start	1.32	1.32	1.38
5 months after pulse start	1.32	1.33	1.40
6 months after pulse start	1.32	1.33	1.41
7 months after pulse start	1.32	1.33	1.43
8 months after pulse start	1.32	1.33	1.45

The estimated bone lead level after 8 months of exposure to 10 ppb WLL is essentially identical to that of baseline; the estimated bone lead level after 8 months of exposure to 100 ppb WLL is higher than baseline but is within the variability of "background" bone lead levels in adults. The marginal bone lead increase at 100 ppb WLL is trivial both clinically and from a causation standpoint. In short, the AALM model used by class plaintiffs indicates that the WLLs present in a vast majority of Flint homes during Feb 10, 2015 to October 2015 did not result in a significant increase in bone lead levels; this exercise proves that Flint tap exposures failed to meet Dr. Hu's preferred criterion for exposure and causation and that plaintiff's answer to the Issue #3 question must be "no".

The AALM model predicts that adult Flint bone lead levels that might have resulted from 8 months of tap WLL exposure are far lower than those measured in the cohorts Dr. Hu cited to in his declaration

As noted above, in his declaration, Dr. Hu cited to several papers that he believes are evidence of general causation for lead exposure and various health effects. Dr. Hu only discussed the BLLs that were presented for the exposed cohorts, if at all; yet several of these studies also or exclusively presented measured bone lead levels from the cohorts (Farooqui et al., 2017; Peters et al., 2007; Rhodes et al., 2003; Tsaih et al., 2004; Weuve et al., 2009; Wu et al., 2003; Zheutlin et al., 2018). The average bone lead levels in all but one of these studies was higher than 20 ug/g, and all studies reported mean bone lead levels greater than 10 ug/g. In comparison, the AALM-modeled bone lead levels reported above for a 60-year-old female after 8 months of exposure to elevated WLLs of 10 and 100 ppb were far lower (below 1.5 ug/g). As such, the bone lead levels reported in the studies cited by Dr. Hu are not relevant to the Flint residential population during February 10, 2015 to October 16, 2015.

Dr. Hu does not cite to any studies that involve lead exposure through tap water ingestion.

Tap water ingestion is not cited as an exposure pathway in any of the studies cited by Dr. Hu. This is consistent with the fact that, as previously discussed in Opinion #1, tap water ingestion is not a major source contributor to adult BLLs. There is no evidence to indicate that the alleged health effects have occurred in any population via tap water ingestion.

Opinion 5: Dr. Michaels' claims of skin rashes and hair loss due to tap water exposure from February 10, 2015 to October 16, 2015 are not supported by the scientific evidence.

Brief overview of Dr. Michaels' findings and conclusions

According to his declaration, Dr. Michaels reviewed the Unified Coordination Group (UCG) *Flint Rash Investigation* report, as well as other literature, and concluded that there was a high degree of certainty supporting general causation between exposure to Flint tap water during the switchover period and skin rash and hair loss outcomes (Michaels Declaration 10/18/22: p. 3). He reviewed the nine Bradford Hill criteria and concluded that the weight of evidence supported a general causation hypothesis associating Flint tap water with skin rashes and hair loss (Michaels Declaration 10/18/22: p. 11-20).

Earlier in this report, when discussing potential systemic health effects related to lead, I mentioned that in order to answer the question posed by Issue #3 ("Were the contaminated water conditions capable of causing harm to Flint residents?"), it is necessary to compare Flint tap WLL doses (during February 10, 2015 to October 16, 2015) to minimum threshold values for each alleged health effect. The same is true for tap water pH, water hardness, and chlorine relative to claims of rashes and hair loss. At no point in his report does report does Dr. Michaels describe the minimum threshold values at which he believes an adverse response would occur, nor does he compare the water quality values that he believes existed in Flint (during February 10, 2015 to October 16, 2015) to any minimum threshold values over any given time frame. Therefore, he has failed to address Issue #3.

It is worth noting that Dr. Michaels acknowledged that ATSDR (2019) found no epidemiology studies of adverse dermal effects associated with chronic lead exposures in his report concerning the first round of child plaintiffs (Michaels Levy 8/6/2020: p. 85; Michaels Napoli 8/6/2020: p. 54).

UCG report findings

In 2016, several months after Flint returned to the use of Lake Huron water, the UCG—a collaboration between local, state, and federal health and environmental agencies—evaluated whether Flint municipal water might be associated with rashes reported by some community residents (UCG 2016: p. 7). The objective of the UCG investigation was to better understand and characterize reported rash cases, explore possible causes of the reported rashes and possible associations with the Flint water supply, and to make recommendations for interventions (UCG 2016: p. 7).

The UCG reviewed the hardness (measured as calcium carbonate concentration), pH, and chlorine concentrations of Flint municipal water at the Flint water treatment plant during the switchover period to determine "whether Flint water might be associated with the rashes experienced by some community residents" (UCG 2016: p. 7). According to their report, during the switchover period, water hardness increased from 98 mg calcium carbonate per liter ($CaCO_3$/L) to 173 mg $CaCO_3$/L,

with “monthly spikes over 250 mg $CaCO_3$/L” (UCG 2016: p. 29). Similarly, pH increased from 7.3 to an average of 7.7, with “monthly spikes of pH over 8.5” (UCG 2016: p. 29). Free chlorine leaving the Flint treatment plant increased in concentration from an average of 0.9 mg/L to an average of 2.0 mg/L and exceeded 3.0 mg/L for “several months” during the summer and fall of 2014 (UCG 2016: p. 29). These data are based on their review of historical water quality data reported by the Flint water treatment plant. These samples were collected as the water left the treatment plant and entered the Flint distribution system and were not collected in the homes of Flint residents (UCG 2016: p. 28-29). It was noted in the UCG report that they “cannot be certain that the measurements at the water distribution system and the conditions at individuals’ residents were the same” but that these measurements “likely...reflect [the] variability in these levels at residents’ homes” (UCG 2016: p. 29). This comment is consistent with my views stated in Opinion #1 regarding variability in the very factors that dictate a person’s exposure to compounds in tap water. Water quality parameters are expected to vary within and between cities, and this variability does not necessarily imply adverse health outcomes for consumers. The significant degree of variability in tap water pH, chlorine, and water hardness amongst the numerous Flint residences during February 10, 2015 to October 16, 2015 makes it difficult if not impossible to treat all class members as a single group with respect to potential risks of skin rashes.

In addition, between January and May 2016, UCG identified 429 residents with rashes following physician referrals and self-nomination. A questionnaire and telephone survey were administered to 390 Flint residents who reported rash and water quality concerns, clinical dermatologic evaluations were performed on 122 resident volunteers, and contemporaneous (2016) water quality testing was performed at the homes of 170 survey respondents. The authors documented 390 rash and 175 hair loss complaints (UCG 2016: p. 9).

UCG reported that 48.5% of their study participants had rash onset prior to October 2015 that worsened during the switchover and 38.2% of respondents had rash onset in October 2015 or later, 57% of whom had an onset in January or February 2016 (UCG 2016: p. 22-23). 13.3% of participants did not report when their rash began, and those who reported rash prior to October 2015 also did not provide a specific date of onset. It is therefore unclear what portion of participants first experienced rash during the Flint switchover and what portion had rash symptoms as a pre-existing condition.

The UCG concluded that “[n]o specific contaminant or group of contaminants in the water samples collected [during the 2016 UCG study] suggest a primary causal factor associated with rashes” (UCG 2016: p. 9). It was noted that water quality parameters (elevated chlorine, pH, and water hardness) “could have played a role in the development on skin irritation or rashes among some participants” but that a consistent pattern between rashes and current water quality was not detected (UCG 2016: p. 9).

The UCG was very clear about the limitations of their study and how their findings should (and should not) be interpreted. For example, their report notes that its findings are “not generalizable to the larger Flint population since the study population was self-selected instead of randomized” (UCG 2016: p. 10). The authors emphasize that, due to the lack of historical data on rash patterns among Flint residents as well as resident-specific historical water samples, it is “impossible” to draw

"definitive" (i.e. causal) conclusions from their research (UCG 2016: p. 10). Further, they note that "studies of the association between tap water quality and skin irritation are generally lacking" (UCG 2016: p. 15). They acknowledge that "[c]orrectly diagnosing rashes is complicated" since the cause is "often multifactorial" (UCG 2016: p. 7). The authors report that atopic dermatitis is the "most common of all chronic childhood diseases," and is experienced by 20% of school-aged children, 13% of adults in Michigan, and up to 30% of people overall in the U.S. (UCG 2016: p. 13). They suggest that their "results should be interpreted with caution and in keeping with the study objectives" (UCG 2016: p. 34).

As described below, Dr. Michaels ignored UCG's cautionary comments and attempted to "re-interpret" the data his own way to support his opinions.

Dr. Michaels' attempt to "re-interpret" the UCG study findings is flawed

In many areas of his report, Dr. Michaels attempts to extend the significance of the UCG findings beyond the authors' intended use. For example, UCG did not conduct any statistical comparisons between groups. When Dr. Michaels was asked by counsel in his deposition whether he agreed that the UCG report does not offer any statistically significant findings linking tap water to adverse health outcomes, Dr. Michaels disagreed, and opined that the changes in water parameters during the Flint water crisis were "not, you know, minor changes that you would have to do a statistical test to verify"; he testified that he believed that the "most reasonable interpretation" of the UCG report was that "these things were not only statistically significant but biologically significant" (Michaels 11/22/22: p. 75, l. 11-13, 18-21). Obviously, one cannot infer statistical or biological significance by "eyeballing the data". Dr. Michaels is attempting to assign significance to the findings of a study that he was never involved with, nor was he asked to participate in interpreting their data or writing the report.

Dr. Michaels did not conduct a proper causation analysis

Dr. Michaels applies each of the nine Bradford Hill criteria to the UCG 2016 report findings and concludes that "the weight of evidence clearly [is] in support of the general causation hypothesis associating corrosive Flint municipal water conditions with skin rashes, hair loss, and other skin conditions" (Michaels Report: p. 11-12). However, Bradford Hill criteria are meant to be analyzed with the sum of evidence from the body of literature on a topic, not a single study. When asked during his deposition if it was appropriate to conduct a Bradford Hill analysis on a single study, plaintiff expert Dr. Hu stated "that would be a misapplication or misunderstanding" of the Bradford Hill criteria (Hu 11/21/22: p. 51, l. 22-23). A full critique of Dr. Michaels' misapplication of the Bradford Hill criteria is available in Section 4.11 of the expert report of Dr. Stacey Benson.

Contrary to Dr. Michaels' claim, the UCG findings do not support a finding of a "biological gradient" for rashes in the Flint population nor did the UCG study report a "strong association" between the development of rashes and exposure to Flint tap water during the switchover

In his report, Dr. Michaels stated that the strength of association between skin rashes and Flint water conditions is "evidently high" based on the UCG findings. Similarly, he concluded that while

UCG's findings "were inadequate to quantify a dose-response relationship," a "biological gradient" could be "inferred qualitatively" because more respondents reported rash onset dates before October 2015 (56%) compared with after October 2015 (44%).

However, Dr. Michaels erred when referring to these percentages as evidence of a "biological gradient" because he gave equal weight to the number of residents with rash onset over the longer, undefined period prior to October 2015 (no start defined start date), and the shorter, defined period between October 2015 and May 2016. Inherent to the differing lengths of these timeframes, it is *expected* that a higher percentage of residents in the "before October 2015" group would develop rashes than residents in the "after October 2015" group.

Further, the UCG did not report any residential tap water quality data collected before or during the switchover; the UCG residential water quality sampling data were limited to the post switchover period between January and May 2016. In the absence of residential tap water quality data before or during the switchover, a biological gradient or dose response relationship between rash onset and water quality cannot be determined. Similarly, UCG did not present any pre-switchover data on rash incidence in Flint; without this "baseline" control group, it is not possible to determine if there was a significant, water-related increase in rashes among Flint residents during the switchover. This was explained in the UCG report: "the lack of historical data on either rash pattern in the community or residence specific water samples from that time period make drawing more definitive conclusions impossible" (UCG 2016: p. 38).

Finally, UCG reported that, despite a range of severity in rash response in individuals who developed rashes post switchover, the post-switchover water quality indicated that chlorine was "not above levels that would cause irritation," water hardness levels were "essentially the same" as those found in Detroit, all but one pH reading was within the "standard range," and disinfection byproducts were below regulatory limits and health-based screening levels (UCG 2016: p. 25-26, 28). Regarding metals, UCG found that "although there were higher levels of some metals in the water of some residents, there were not generally high levels across all homes with reported rashes" (UCG 2016: p. 25). Regarding rash outcomes, following clinical evaluations, the dermatologists were "unable to definitely state that any skin condition was causally associated with the water" and "they concluded that the spectrum and severity of rashes among study participants was similar to that seen in their daily practices for the same time of year" (UCG 2016: p. 31, 33).

In short, 1) the water quality parameters were too low post-switchover to cause rashes, yet several individuals claimed they developed rashes post-switchover (i.e., non-tap sources caused rashes), 2) there was no relationship between water quality and incidence or severity of rashes in the post-switchover group, and 3) and upon clinical examination, the rashes observed were expected given the time of year. These findings directly contradict any claim of a biological gradient or dose response relationship between water quality and rashes.

Contrary to Dr. Michaels' claim, the evidence indicates that metals are not responsible for rashes in Flint residents

Dr. Michaels reported that "metals [are] known to be associated with causation of allergic contact dermatitis, such as arsenic, chromium, cobalt, copper, nickel, silver, thallium, and zinc (Michaels 10/28/22: p. 17)." He further stated that "the role of metals in inducing skin rashes specifically among Flint residents during the Flint Water Crisis was documented by the UCG (2016)" (Michaels 10/28/22: p. 17).

On the contrary, the UCG report clearly states that tap water metals measured in 2016 were not responsible for rashes in Flint residents. Metals associated with allergic or irritant contact dermatitis (arsenic, total chromium, silver) were detected at concentrations "below the lowest levels specified by drinking water regulations for all samples from all homes" and the levels of nickel detected were "well below the levels thought to elicit skin sensitization and reaction" or dermatitis (UCG 2016: p. 8, 35). Metal levels "were not [at] generally high levels across all homes with reported rashes" and the levels of metals and minerals are also "generally similar" to those reported in water for the City of Chicago (UCG 2016 p. 25). Additionally, "[n]one of the levels of these metals were considered by participating dermatologists to be sufficiently high enough to be clinically significant or to cause rashes" (UCG 2016 p. 35). While some metals are associated with allergic contact dermatitis, the exposures experienced in Flint were too low to elicit that response.

There is no evidence that hair loss results from rashes on the scalp

Dr. Michaels claimed that the UCG report "confirms...direct and/or indirect causation of hair loss" (Michaels 10/28/2022: p. 4). He explained that chronic exposure to Flint water was "capable of causing (at least temporary) hair loss as a secondary effect of long-duration scratching and scalp irritation" (Michaels 10/28/2022: p. 4). Citing to the UCG report data, he also noted that "[c]orrosive water conditions also might have a primary relationship with (at least temporary) hair loss, if elevated water pH, chlorine, and hardness can be verified as causes of hair brittleness and breakage" (Michaels 10/28/2022: p. 5).

Dr. Michaels provides no citations as evidence that increased scalp scratching caused hair loss among Flint residents. His opinions are speculation and not founded on scientific research. In addition, there is no reason to believe that the water conditions observed during or after the Flint switchover were capable of causing hair loss. In direct contradiction to Dr. Michaels' claim, the UCG report concluded that "[t]here was no specific contaminant, nor group of contaminants, in current water samples taken that suggest a primary causal factor associated with the occurrence of reported skin conditions or hair loss" (UCG 2016: p. 37). They noted that "[h]air loss has many possible causes that present in different but often very distinct patterns" and specifically hair loss by the roots is more likely to be related to internal factors (UCG 2016: p. 14).

The available data collected by UCG in 2016 also suggests no association between water exposure and hair loss. While 175 respondents reported hair loss during their initial interview, only 14 out of 122 respondents reported hair loss during their clinical evaluations and 7 of these 14 cases were "deemed to be definitely unrelated to contact with the Flint water" (UCG 2016: p. 32). The hair loss

of the remaining 7 respondents was classified as “possibly related” because their condition had no known cause (UCG 2016: p. 32). However, the 7 respondents with hair loss ‘possibly related’ to water exposure all had other conditions that could explain their hair loss: 4 had autoimmune alopecia, 2 had a stress-related temporary shedding condition, and 1 had eczema with scalp involvement (UCG 2016: p. 32). The hair loss experienced by these respondents does not suggest that environmental exposure factors were responsible.

The published weight of evidence does not indicate a causal relationship between exposure to Flint municipal water and rash outcomes among Flint residents

The UCG report noted that “studies of the association between tap water quality and skin irritation are generally lacking” (UCG 2016: p. 15). This conclusion is consistent with observations by others. For example, a comprehensive review of research on water quality and skin irritation found that there was “insufficient evidence to evaluate the effects of domestic tap water, and its chemical constituents or parameters, on skin irritation in humans” (WRc, 2011: p. 1). Similarly, a meta-analysis on the relationship between exposure to hard water and the risk of developing atopic eczema concluded that there was a “lack of high-quality evidence” on the effect of water hardness and atopic eczema in adults (Jabbar-Lopez et al., 2021).

It was suggested in the UCG report that, based on the limited available evidence, it is possible that elevated measures of hard water, alkalinity, and chlorine are *associated* with skin dryness and irritation generally (not specific to any age group) (UCG 2016: p. 29). As described below, I have reviewed the references cited by UCG to support those observations, as well as additional published research, and have determined that these results do not support a conclusion that elevated water hardness, pH, and/or chlorine *caused* skin irritation in Flint adolescents or adults during the switchover period.

Review of literature cited by UCG

As noted earlier, the multi-defendant Flint class consists of residents who were at least 18 years old as of the date of the class notice, which was August 17, 2022. Hence, anyone who had turned 18 by the date of the class notice would have been a little over 10 years old by February 10, 2015. This means that skin rash studies that consider only infants or children less than 10 years of age are not relevant to this case. Infants in particular are a unique subpopulation because the human dermal barrier is not fully developed until the age of approximately one year old. Infants are generally more susceptible to skin conditions such as atopic eczema or atopic dermatitis.

None of the studies cited in the UCG report actually evaluated skin conditions in individuals ≥ 10 year of age; most of the cited studies evaluated infants or groups of children that were generally < 10 years of age. Hence, a majority of the studies and results cited by UCG are simply not relevant to claims of rashes in Flint adolescents and adults. Further, the water quality parameters evaluated in these studies generally far exceed what those reported in Flint during the switchover.

Hard Water

Regarding water hardness, the UCG report cited McNally et al. (1998), Miyake et al. (2004), and Perkin et al. (2016) as evidence that exposure to hard water is associated with skin dryness and irritation. Perkin et al. (2016) evaluated over 1,300 three-month-old infants in the United Kingdom who were examined for atopic dermatitis. $CaCO_3$ concentrations were measured in local domestic water supplies and ranged from 3 to 490 mg/L. Infants exposed to "high" concentrations of water hardness (exact concentration not reported) had significantly increased risk of atopic eczema. However, as noted above, these findings cannot be considered relevant to the Flint class (adolescents and adults).

McNally et al. (1998) evaluated parent-reported atopic eczema among over 4,000 children aged 4 to 11 and over 3,000 children aged 11 to 16 in the United Kingdom. Water hardness was determined by total concentration (mg/L) of calcium and magnesium salts and divided into four categories: 1) 118-135 mg/L salts, 2) 151-157 mg/L salts, 3) 172-215 mg/L, 4) 231-314 mg/L. In the 4 to 11-year-old children, the prevalence of atopic eczema was significantly increased only at the highest concentration (231-314 mg/L) and in the adolescents there was no increase in atopic eczema at any of the water hardness concentrations. Hence, this study does support a conclusion that increased water hardness does not cause atopic eczema in adolescents or adults. These concentrations of calcium and magnesium salts cannot be directly compared to the calcium carbonate ($CaCO_3$) concentrations measured during the Flint switchover.

Miyake et al. (2004) performed a study on 6- to 12-year-old children living in Japan with medical histories of clinically diagnosed atopic dermatitis. Children exposed to estimated $CaCO_3$ levels greater than 76 mg/L had a small (1.5%) increase in the prevalence of atopic dermatitis compared to children exposed to $CaCO_3$ levels below 48 mg/L. The authors noted that more information was needed “to draw a conclusion regarding...water hardness as an independent risk factor” for atopic dermatitis and noted that environmental and genetic factors may contribute to this relationship (Miyake et al., 2004: p. 37). This study also supports the conclusion that increased water hardness alone does not cause atopic dermatitis in adolescents.

In 2011, the Drinking Water Inspectorate—a division of the Department for Environment, Food, and Rural Affairs that regulates public water supply companies in England and Wales—published a literature review of evidence of tap water quality and usage and skin irritation and sensitization or “any related health effects” (WRc, 2011: p. 1). They noted that there was a perceived association between tap water exposure and skin disease that was perpetuated by non-scientific literature, but that, at the time, this theory had not been substantiated by a review of the scientific literature (WRc, 2011: p. 5). The authors reviewed literature addressing 502 chemicals and chemical parameters in drinking water and health endpoints of skin irritation, eye irritation, respiratory and gastrointestinal tract irritation, and sensitization potential (WRc, 2011: p. 25).

With respect to water hardness, the authors reviewed the aforementioned studies by McNally et al. (1998) and Miyake et al. (2004), well as a study by Arnedo-Pena et al. (2007). They determined that these studies “do not rule out the possibility that another environmental factor, with a similar spatial distribution to water hardness, accounts for the observed association” (WRc, 2011: p. 54). The

authors noted that a "number of observational studies suggest that regular exposure of the skin to hard water, as opposed to soft water, exacerbates and/or prolongs the duration" of atopic dermatitis, but again concluded that there was an absence of "robust" clinical studies that evaluate the effects of hard water on the skin and that further investigation is needed (WRc, 2011: p. 57, 72). They noted that "these observations alone are insufficient to draw any firm conclusions other than the need for further research" (WRc, 2011: p. 55).

Water pH

The aforementioned WRc Group study noted that, in animal studies, skin damage occurred following exposure to highly alkaline solutions with a pH of 10, and skin disorders were exacerbated following exposure to pH values greater than 11 (WRc, 2011: p. 47). However, as noted previously, the water pH during the switchover period averaged 7.7 with spikes below 10; the exposures observed in Flint are therefore below alkalinities associated with skin damage in animals (UCG 2016: p. 29). Regarding human studies, the WRc Group cited to the same studies on hard water discussed above and noted that the alkalinity of water is a parameter that is affected by natural water hardness. They found that "[s]pecific conclusions about the effect of individual chemical properties of tap water cannot be drawn from these studies" and did not report any direct association between tap water pH and human skin irritation among those of similar age to the Flint class (WRc, 2011: p. 15, 54-55, 76).

Chlorine

Perkin et al. 2016 was cited in the UCG report as evidence that elevated total chlorine concentrations are associated with skin dryness and irritation (UCG 2016: p. 29). However as noted earlier, this study only evaluated infants and therefore it is not relevant to the Flint class of adolescents and adults. Furthermore, Perkin et al. concluded that whether chlorine contributes to the development of atopic dermatitis in infants "remains uncertain" (Perkin et al., 2016: p. 516). Hence, this study obviously does not support the conclusion that elevated chlorine levels during the switchover period resulted in skin rashes in Flint adolescents and adults.

While not discussed within the UCG report, the study by McNally et al. (1998) mentioned above also describe dermal exposure to chlorinated water (concentration not reported). McNally et al. (1998)McNally et al. found there was no significant association between chlorine exposure and eczema among 11- to 16-year-olds. This study supports the conclusion that exposure to elevated chlorine would not cause skin irritation among the Flint class.

Summary

A review of the cited studies in the UCG report indicates that very few of the findings are actually relevant or applicable to the Flint class because the studies mostly evaluated infants and children less than 10 years of age, and the one study that actually did evaluate adolescents (water hardness and chlorine in McNally et al.) found no association. Overall, the studies cited in the UCG report support the conclusion that elevated water hardness, pH, or chlorine concentrations are not associated with an increased incidence of skin rashes in adolescents or adults, and they certainly

support the conclusion that the water parameter concentrations observed during the switchover were too low to cause skin rashes or any other health effects in the Flint class.

Additional studies not cited by UCG

I evaluated the published literature to identify additional studies of the potential association between exposure to hard water, alkalinity, and/or elevated chlorine levels and skin irritation. While there are several published studies that found no association between these water quality parameters and skin responses in infants and young children, I did not include these studies and instead specifically focused on studies that included adolescents and adults. I find that the weight of the available evidence does not suggest a causal relationship between these water quality parameters and skin irritation in Flint adolescents and adults.

Hard Water

Arnedo-Pena et al. (2007) (which was cited in the aforementioned WRc report) evaluated the association between exposure to residential hard water and the prevalence of atopic eczema among children aged 13 to 14 years old in Spain. Three categories of water hardness were defined as $CaCO_3$ concentrations of <200 mg/L, 200-250 mg/L, and >300 mg/L. The authors found no association between water hardness and prevalence of atopic eczema. The concentrations observed during the Flint switchover (< 300 mg/L) similarly are not associated with the prevalence of atopic eczema.

Thomas et al. (2011) measured the effect of softening water on existing eczema among children aged 6 months to 16 years. At baseline, all participants had residential water hardness equal to or exceeding 200 mg $CaCO_3$ /L. Participants were randomized into two groups: one received an ion-exchange water softener in addition to usual eczema care, and the second only received usual eczema care. The authors concluded that the use of water softener did not significantly improve eczema outcomes.

Water pH

There is limited research on the association between water pH and skin irritation and the majority of studies on this topic instead focus on the pH of the skin surface in relation to dermal effects. However, water pH in other cities where increased skin irritation is not reported can serve as a reference for acceptable exposures. For example, the city of Denver historically delivered water with a pH between 7.5 to 8.5, with a target pH of 7.8. In 2020, the city increased their target pH to 8.8, with an achieved range of 8.5 to 9.2 (DenverWater). Similarly, the pH of tap water in Washington, D.C. ranges from 7.4 to 8.0, in Seattle ranges from 7.5 to 8.5, and in Kansas City ranges from 9.5 to 10.3 (DCWater, 2017; KCWater, 2020; SPU, 2016). There have not been reports of increased skin irritation associated with exposure to water in these cities. The water pH observed in Flint during the switchover (average: 7.7; maximum < 10) is similarly not expected to cause skin irritation.

Chlorine

One additional study that evaluated exposure to chlorine and dermal outcomes in adults (aged 18 to 60 years) was identified and a clear effect with chlorine was not observed (Danby et al., 2018). However, this study was designed to observe the effect of hand washing using a surfactant, and may not be directly relevant to the Flint class. Regardless, these findings align with the conclusions from the studies cited by UCG (described above) which find no association between exposures to chlorine at the concentrations observed during the switchover and skin irritation among adolescents and adults.

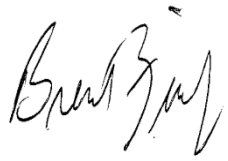
Summary

In summary, the literature regarding incidence of skin rashes in adolescents and adults following exposure to hard water, alkaline water, and/or elevated chlorine levels and skin irritation is sparse but does not suggest that increases in any of these water quality parameters causes skin irritation. The majority of the available literature evaluates exposures in excess of the levels measured at the Flint water treatment plant during the Flint switchover and finds no association with dermal irritation outcomes.

V. CLOSING COMMENTS

I submit these opinions to a reasonable degree of scientific certainty and am prepared to support them in both deposition and/or courtroom testimony. I may supplement this report if additional information becomes available or I am asked to address other issues.

Respectfully,



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2/3/2023
Date

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ATTACHMENT A

Brent L. Finley, PhD, Toxicology
Senior Managing Principal Health Scientist

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Summary of Experience

Dr. Brent Finley has over 30 years of experience conducting and managing studies involving chemical exposures and human health risk assessment. He specializes in applied research, litigation support, and risk-based site investigations. Dr. Finley has studied the health effects of exposure to a wide range of chemicals and consumer products, including asbestos, artificial flavorings, and metal hip implants. He has provided expert witness testimony in lawsuits involving alleged health risks associated with consumer exposures to asbestos in cosmetic talc, occupational exposure to flavorings, and dioxins in foods. Dr. Finley has been involved in the preparation of more than 400 risk assessments and has published over 160 peer-reviewed papers on the topic of health risk assessment.

Education and Degrees Earned

- PhD, Toxicology/ Pharmacology, Washington State University, 1986
- BA, Biological Sciences, Cornell University, 1982

Certifications

- Diplomate, American Board of Toxicology (1991)

Professional Honors/Awards

- Best Published Paper Regarding Medical Devices. 2016. A preliminary evaluation of immune stimulation following exposure to metal particles and ions using the mouse popliteal lymph node assay. Medical Device and Combination Product

Specialty Section – Society of Toxicology Annual Meeting, Baltimore, MD, March 2017.

- Recipient of American Industrial Hygiene Conference & Expo (AIHce)
  - "Best of Session" Poster Award. Session 403. 2016 - Characterization of Naturally Occurring Airborne Diacetyl Concentrations Associated with the Preparation and Consumption of Unflavored Coffee
- American Industrial Hygiene Conference & Expo (AIHce)
  - "Best of Session" Poster Award. Session 403. 2015 - Pilot Study of Exposures to Airborne Concentrations of Naturally Occurring Diacetyl During Coffee Consumption.
- Best Published Paper Regarding Medical Devices. 2014 - Toxicology-based cancer causation analysis of CoCr-containing hip implants: a quantitative assessment of genotoxicity and tumorigenicity studies. Medical Device and Combination Product Specialty Section - Society of Toxicology Annual Meeting, San Diego, CA, March 2015.
- American Industrial Hygiene Conference & Expo (AIHce)
  - "Best of Session" Poster Award. Session 403. 2014 - Diacetyl and 2,3-Pentanedione Exposures Associated with Cigarette Smoking: Implications for Risk Assessment of Food and Flavoring Workers.
- American Industrial Hygiene Conference & Expo (AIHce)
  - "Best of Session" Poster Award. Session 401. 2013 - A Comparison of Reported Peak Concentrations of Diacetyl in the Workplace and Estimated Diacetyl Concentrations in Mainstream Cigarette Smoke.
- American Industrial Hygiene Conference & Expo (AIHce)
  - "Best of Session" Poster Award. Session 403. 2013 - A Comparison of Workday-Duration Occupational Exposures to Diacetyl to Estimated Exposures Associated with Cigarette Smoking
- American Industrial Hygiene Association's Exposure Assessment Strategies
  - "Best of Session" Poster Award, 2012 - Analysis of Time-Weighted Average Concentrations of Formaldehyde During Keratin Hair Smoothing Treatments
- American Industrial Hygiene Association's Indoor Environmental Quality (IEQ)
  - Committee's "Best IEQ Paper Award", 2011 - Characterization of Formaldehyde Exposure Resulting from the Use of Four Professional Hair Straightening Products
- Recipient of Society of Toxicology's "Outstanding Published Paper Award", 1995
- Recipient of Society of Toxicology's "Graduate Student Best Paper" Award, 3rd place, 1985
- Recipient of Pharmaceutical Manufacturer's Association Research Grant, 1982

Key Projects (Partial List)

Specific Experience with Pharmaceuticals, Medical Devices and Consumer Products

Conducted and published extensive research related to the potential risks of cobalt and chromium exposure from metal-containing hip implants.

Analyzed the safety and various failure modes associated with metal-on-metal and other types of hip implants.

Awarded Best Published Paper Regarding Medical Devices at Society of Toxicology Annual Meeting in 2014 and 2016 for papers related to metal-on-metal hip implant toxicology.

Currently serving as an expert witness in exposure assessment, industrial hygiene, and toxicology in cases involving popcorn worker exposure to diacetyl and artificial butter.

Evaluated the metabolic and toxicity results (from animal and clinical data) of Levaquin and Oflaxacin to determine whether and to what degree the two drugs confer a different therapeutic index.

Consulted in personal injury lawsuits involving healthcare worker use of latex gloves and associated claims of dermal sensitization.

Prepared an expert report regarding likelihood of association between use of rug cleaner and plaintiff's alleged symptoms of RADS

Prepared a report and manuscript describing the possible health risks associated with exposure to rubber tire particles in the environment

Served as an expert exposure and risk assessment expert in a case involving formaldehyde emissions from kitchen cabinets.

Deposed in a case involving claims of health harm associated with exposure to airbag contents (azide and talc) following airbag deployment and failure.

Conducted an analysis of health risks associated with exposure to lead in detailing paint that contained up to 30% lead.

Specific Experience with Benzene

Served as an expert in exposure and risk assessment in a case involving exposure to benzene in coal tar pitch volatiles in roofing tar

On behalf of a pipeline manufacturer, evaluated health claims related to household exposure to benzene vapors following an underground leak

Deposed in a case involving alleged exposures to benzene in a degreasing solvent

Conducted over 30 environmental risk assessments in which benzene in groundwater was a significant contributor to potential human health risks

Served as expert witness for Oryx Energy in a case involving property damage/health harm from a groundwater benzene plume

Specific Experience with Groundwater Risk Assessment

Numerous evaluations of MTBE fate/transport and potential health risks associated with leaking underground storage tanks.

Involved in several cases (Aerojet, CNH) in which current and historical residential exposure to chlorinated solvents in groundwater and tap water is alleged. These cases involve exposure and dose reconstruction (tap water ingestion and inhalation and dermal exposures during showering) using measured and modeled data, and conclusions concerning alternative causation.

Assessed exposure to Cr(VI) in groundwater via the use of simulation studies (all subsequently published in the peer-reviewed literature) and served as a testifying expert in a case involving numerous plaintiffs in Hinkley, CA

Testified at a hearing regarding the adequacy of several atrazine databases for use in health risk assessment.

Specific Experience with Asbestos

Serving as a consultant and expert witness in a series of cases involving alleged exposure to asbestos in welding rods, joint compound, and automotive and crane brake linings, clutches, and gaskets.

Deposed in a premise case involving bystander exposure to asbestos-containing insulation.

Published eleven papers in the peer-reviewed literature pertaining to asbestos exposure, warnings, and health risks from a state-of-the art perspective.

Specific Experience with Chlorinated VOCs

Deposed as an expert witness in a toxic tort case involving alleged exposure to numerous chlorinated solvents (TCE, TCA and CCI4) in groundwater (Comeaux v. Conde Vista). Dr. Finley reviewed the existing groundwater and soil gas data and determined, based on site-specific vapor migration modeling and an evaluation of household exposure pathways, that any inhalation exposure to chlorinated solvents that might have occurred was insufficient to cause the plaintiffs claimed health effects.

Assisted defense counsel in litigation involving alleged residential TCE exposure in the 1950s and 1960s (Stuart v. Lockheed).

Served as a panel member for the International Life Sciences Institute Working Group on estimation of dermal and inhalation exposure to chlorinated contaminants (TCE, TCA, perchloroethylene) in drinking water.

Chaired an expert panel review of EPA's inhalation toxicity criteria for 1,2-dichloroethane and 1,l,2-trichloroethane.

Conducted and published a probabilistic analysis of household exposure to TCE and perchloroethylene in tap water to demonstrate the health protectiveness of the EPA's MCLs.

Specific Experience with Stack Emissions

Prepared a multi-pathway exposure and risk assessment for the U.S. Army chemical weapons incinerator in Tooele, Utah. The purpose of the analysis was to assess the merits of an injunction filed by a plaintiff's consortium that operating the incinerator would pose a risk to surrounding residents. Testified twice in court on behalf of the Department of Justice. The judge's decision to permit the incinerator to operate was based in part on Dr. Finley's findings and testimony.

Served as an expert exposure and risk assessment expert in a case involving community exposures to particulate emissions from a steel-manufacturing facility.

Prepared a multi-pathway assessment of the health risks associated with emissions of dioxin particulates and vapors from a combustion source. The State of California had previously determined that the dioxin risks were significant, and that the client would have to warn the surrounding residential community. Used refined risk assessment techniques to demonstrate that the potential health risks were insignificant. Exposure pathways considered included ingestion of mother's milk, vegetable crops, and local meat and dairy products.

Prepared a multi-pathway exposure and risk assessment for a state Superfund site in Illinois. The assessment demonstrated that particulate emissions from a client's insecticide-formulating facility did not pose a significant health risk to the surrounding community. Accordingly, the Illinois EPA issued a no-action alternative for the site and no remediation was required.

Specific Experience with Dioxins and PCBs

Expert witness in a case concerning alleged "recreational" exposures to Aroclor 1254 in soil at a former capacitor manufacturing site.

Involved in research with EPA and WHO scientists to refine the current TEF scheme for dioxin and PCB risk assessment.

Conducted (and published) a human exposure study designed to assess the degree to which naturally occurring dietary compounds ("endodioxins") contribute to the overall TEQ dose in the general population.

Conducted (and published) the first soil bioavailability study to examine all 17 2,3,7,8-substituted PCDD/Fs.

Evaluated dioxin levels in the blood of workers employed at magnesium facility in Utah.

Served as principal-in-charge of a Superfund project involving dioxin- and PCB-contaminated sediments in a major waterway in New Jersey. This project involves the use of fingerprinting techniques for source identification, food-web modeling and defense against natural resource damage claims. Dr. Finley has published over 25 papers regarding the proper use of exposure and risk assessment techniques for this site.

Designed the first sediment toxicity study conducted to demonstrate that dioxin poses no risk to benthic invertebrates.

Assisted in the development, conduct, and interpretation of a year-long creel-angler survey designed to accurately estimate the fish and crab consumption rates (and associated risks) in a PCB-contaminated waterway.

Conducted (and published) an analysis of human and ecological risks associated with suspension of dioxin-contaminated sediments as a result of dredging.

Invited to speak at an EPA external peer-review workshop for the guidance document "PCBs: Cancer Dose Response Assessment and Application to Environmental Mixtures."

Evaluated PCB-related risks to anglers consuming fish from the Fox River, Wisconsin.

Specific Experience with Chromium

Designed and conducted the first human sweat extraction study for the purposes of measuring bioavailability of chromium from soil.

Organized and chaired an expert panel investigation into the technical merits of New Jersey Department of Health's urinalysis and house dust analysis of residents living near chromium-impacted areas.

Served as principal-in-charge of an industry-funded investigation into OSHA's basis for lowering the permissible exposure limit for chromium (VI).

Served as an expert exposure and risk assessment witness in a toxic tort case involving residential exposure to chromium emissions from a former plating facility in Southern California.

Managed a complex assessment of the health risks associated with chromium-contaminated soil at more than 100 sites in New Jersey. This assessment entailed developing new sampling and analytical techniques for ambient chromium concentrations, and designing and implementting several human exposure studies and extensive regulatory negotiations with the New Jersey Department of Environmental Protection.

Conducted a $2M human patch-testing study for the purposes of identifying the dermatitis elicitation threshold for chromium. This study was used to demonstrate that the Agency's initial position on dermatitis-based cleanup standards was seriously flawed.

Invited to speak at an EPA Workgroup session on chromium reference doses regarding proposed methods for setting a chromium (III) reference dose.

Prepared and submitted comments to the EPA's proposed inhalation "reference concentrations" for chromium. As a result of the submitted comments, EPA withdrew the proposed values. Invited to serve on an EPA work group to evaluate better methods for setting these criteria.

Specific Experience with Arsenic

Performed an exposure assessment study evaluating impacts of arsenic in groundwater to soil and fish. A conceptual site model was constructed to determine the complex potential exposure conditions that may exist offsite of a manufacturing facility in Indiana under the Indiana Voluntary Remediation Program. Work for this project included conducting an inorganic fingerprinting analysis, which demonstrated that offsite arsenic concentrations were not derived from onsite sources. A background concentration study was conducted to eliminate offsite areas that did not exceed background levels, and a survey of arsenic in fish tissue from the adjacent surface water body indicated that arsenic concentrations in the fish were consistent with background. A health risk assessment was also conducted.

Assisted a group of Florida physicians in drafting a position paper to the Secretary of the Department of Health concerning children's health risks associated with contacting arsenic from CCA-treated playground equipment. Reviewed and summarized the pertinent epidemiologic and other human arsenic toxicological literature and presented to the Secretary the opinion that, based on the available literature, contact with CCA (chromated copper arsenate) treated wood by children under the conditions of typical playground use did not pose an unacceptable health risk.

Evaluated potential health risks associated with fish consumption among recreational and subsistence anglers in the San Diego Bay. Exposure pathways were developed to assess cancer and non-cancer risks associated with consumption of fish and shellfish. Potential chemicals of concern included PCBs, arsenic, copper, cadmium, and mercury.

Specific Experience with Pathogens

Designed a sampling analysis program for combined sewer overflows (CSOs) in the Passaic River. The purpose of the program is to conduct a pathogen risk assessment for recreationists who come in contact with the surface water of the River. Several samples have been collected

during storm events and the bacterial/viral content of the samples is currently being interpreted to develop estimates of increased risk of various diseases.

Interpreted pathogen content of tap water samples following plaintiff's assertion that ingestion had caused an array of autoimmune diseases.

Deposed as an exposure and risk assessment expert in EPA et al v. City of Los Angeles. The EPA alleged that uncontrolled sewage spills had impacted Santa Monica Bay to such a degree that beach recreators were at great risk of developing pathogen-related diseases. Dr. Finley reviewed the monitoring data and developed a pathogen exposure and risk analysis.

Specific Experience with Creosote, PAHs, Coal Tar and Wood-treating Sites

Deposed as an expert exposure and risk assessment witness in a federal case involving creosote and pentachlorophenol production at a former wood-treating site in St. Louis.

Currently serving as an exposure and risk assessment expert in a case involving community allegations of health effects from historical exposures to emissions from a wood-treating facility in Louisiana.

Served on an expert panel that evaluated the health risks associated with creosote and coal tar-containing products; the panel concluded that the toxicology and epidemiology data do not support a conclusion that these substances are carcinogenic in humans.

On behalf of a creosote manufacturer, helped determine "background" levels of PAH exposure in a community (from diet, etc.) vs. PAH exposures associated with trace creosote levels in residential soils.

Conducted over 50 environmental risk assessments (RCRA, CERCLA, RBCA, etc.) in which PAHs in soils were the primary contributor to potential human health risk; a majority of these involved derivation of site-specific soil standards using state-of-the-art exposure assessment techniques. Approximately half of these projects involved regulatory interaction.

Specific Experience with Warnings, Labels and State-of-the-Art Analyses

Evaluated compliance of various hair straightening products with OSHA formaldehyde standards

In the mid-1980's, developed warning language for Material Safety Data Sheets in accordance with the new OSHA Hazard Communication Standard.

State-of-the-art expert in warnings and labels in cases involving worker exposure to asbestos during joint compound use and brake servicing.

Served as a state-of-the-art expert in warnings and labels in cases involving worker exposure to "draw fluids" during automobile manufacture.

Assessed whether the manufacture of a petroleum solvent "had knowledge" that using the solvent might be associated with exposure to significant levels of benzene.

Assisted counsel in determining whether labels and warnings on a lead-containing paint were adequate and appropriate under Proposition 65.

Specific Experience with Coal Ash Metals and Radionuclides

On behalf of defendants, deposed in a case involving claims that coal ash metals (arsenic and hexavalent chromium) and radionuclides had migrated off-site from a series of impoundments into private wells of residents downgradient of the impoundments. I evaluated the on-site groundwater and off-site groundwater and tapwater data and concluded that 1) a large fraction of the metals and radionuclides in the tapwater were naturally occurring and unrelated to the impoundments, and 2) the tapwater constituent levels were far too low to pose a health risk to residents using the water for consumption and other purposes. The case subsequently settled.

On behalf of defendants, deposed in a case in which several thousand tons of coal ash were deposited into a municipal landfill over a period of several years. Claims include property damage and potential health harm from arsenic and other metals. There are also cross claims from other defendants that contributed to materials in the landfill. This case is still ongoing and is planned for trial in Fall 2019.

On behalf of defendants, currently evaluating claims of health risk due to wind erosion of a coal ash aggregate (AGREMAX) this is used as daily landfill cover throughout Puerto Rico. Residents living in the same regions as the landfill locations claim that deposition of ash-related radionuclides and metals onto their properties poses a substantial and imminent health hazard. I am expected to testify if/when these claims go to trial.

Specific Experience with Industrial Slags

Served as principal-in-charge of a risk assessment involving residential exposure to arsenic-containing industrial slag.

Designed and conducted a human exposure study (involving the direct ingestion of soil) for the purposes of assessing the disposition of a priority pollutant metal in an industrial slag.
Served as principal-in-charge of an ongoing evaluation of the public health risk associated with numerous beneficial uses of steel slag.

Other Projects

Evaluating environmental risks associated with potential leaching of metals and PFAS compounds from solar array panels.

Demonstrated that post-remedial concentrations of mercury in soil at an industrial site in Puerto Rico did not pose a significant health risk to individuals working on the property. EPA Region II then formally closed the site with no further remediation required.

Prepared an avian health risk assessment at a state Superfund site. The assessment involved collecting and analyzing live and dead birds for cyclodiene content.

Managed two ecological impact studies in Melbourne, Australia.

Served as principal-in-charge of four separate RCRA risk assessments involving more than 400 solid waste management units.

On behalf of a former toxaphene manufacturer, conducted an analysis of toxaphene-related risks to humans consuming fish in a waterway in Georgia.

PUBLICATIONS/PRESENTATIONS

Peer-Reviewed Publications

1. Ierardi, A.M., C. Mathis, A. Urban, N. Jacobs, **B.L. Finley**, and S.H. Gaffney. 2021. Potential airborne asbestos exposures in dentistry: a comprehensive review and risk assessment. Crit Rev Tox. 51(4): 301-327.

2. Marsh, G.M., A.M. Ierardi, S.M. Benson, and **B.L. Finley**. 2019. Response to letters regarding "Occupational exposures to cosmetic talc and risk of mesothelioma: an updated pooled cohort and statistical power analysis with consideration of latency period." Inhal Tox. Advance online publication, Dec. 18, 2019. doi: 10.1080/08958378.2019.1702744.

3. Jacobs, N.F.B., K.M. Towle, **B.L. Finley**, and S.H. Gaffney. 2019. An updated evaluation of potential health hazards associated with exposures to asbestos-containing drywall accessory products. Crit Rev Tox. Advance online publication Aug. 5, 2019. doi: 10.1080/10408444.2019.1639612.

4. Marsh, G.M., A.M. Ierardi, S.M. Benson, and **B.L. Finley**. 2019. Occupational exposures to cosmetic talc and risk of mesothelioma: An updated pooled cohort and statistical power analysis with consideration of latency period. Inhal Tox. Advance online publication August 5, 2019. doi: 10.1080/08958378.2019.1645768.

5. Beckett, E.M., W.D. Cyrs, A. Abelmann, A.D. Monnot, S.H. Gaffney, and **B.L. Finley**. 2019. Derivation of an occupational exposure limit for diacetyl using dose-response data from chronic animal inhalation exposure study. J Appl Tox. 39(5):688-701

6. Kovochich, M., **B.L. Finley**, R. Novick, A.D. Monnot, E. Donovan, K.M. Unice, E.S. Fung, D. Fung, and D.J. Paustenbach. 2019. Understanding outcomes and toxicological aspects of second generation metal-on-metal hip implants: A state-of-the-art review. Crit Rev Toxicol. Advance online publication, March 26, 2019. doi: 10.1080/10408444.2018.1563048.

7. Barlow, C.A., G.M. Marsh, S. Benson, and **B.L. Finley**. 2018. The mineralogy and epidemiology of cosmetic talc. Tox Appl Pharm. Advance online publication, May 30, 2018. doi: 10.1016/j.taap.2018.05.036.

8. **Finley, B.L.**, S.M. Benson, and G.M. Marsh. 2018. Response to letters regarding "Cosmetic talc as a risk factor for pleural mesothelioma: A weight of evidence evaluation of the epidemiology." Inhal Tox. Advance online publication, Feb. 21, 2018. doi: 10.1080/08958378.2018.143850.

9. Fung, E.S., K.M. Unice, D.J. Paustenbach, **B.L. Finley**, and M. Kovochich. 2018. Methods for sterilizing clinically relevant wear particles isolated from metal-on-metal hip implants. Sci Rep. 8(1):2384.

10. Fung, E.S., A. Monnot, M. Kovochich, K.M. Unice, B.E. Tvermoes, D. Galbraith, **B.L. Finley**, and D.J. Paustenbach. 2017. Characteristics of cobalt-related cardiomyopathy in metal hip implant patients: An evaluation of 15 published reports. Cardiovasc Tox. Advance online publication, Nov. 29, 2017. doi: 10.1007/s12012-017-9433-z.

11. **Finley, B.L**., S.M. Benson, and G.M. Marsh. 2017. Cosmetic talc as a risk factor for pleural mesothelioma: A weight of evidence evaluation of the epidemiology. Inhal Tox. 29(4):179-185.

12. Kovochich, M., E.S. Fung, E. Donovan, K.M. Unice, D.J. Paustenbach, and **B.L. Finley**. 2017. Characterization of wear debris from metal-on-metal hip implants during normal wear versus edge-loading conditions. J Biomed Mat Res B. Advanced online publication, May 8, 2017. doi: 10.1002/jbm.b.33902

13. **Finley, B**., P.K. Scott, M.E. Glynn, D. Paustenbach, E. Donovan, and K.A. Thuett. 2017. Chromium speciation in the blood of metal-on-metal hip implant patients. Tox Environ Chem. 99(1):48-64.

14. Winans, B., B.E. Tvermoes, K.M. Unice, M. Kovochich, E.S. Fung, W.V. Christian, E. Donovan, **B.L. Finley**, I. Kimber, and D.J. Paustenbach. 2016. Data on the histological and immune cell response in the popliteal lymph node in mice following exposure to metal particles and ions. Data in Brief. 9:388-397.

15. Tvermoes, B.E., K.M. Unice, B. Winans, M. Kovochich, W.V. Christian, E. Donovan, E.S. Fung, **B.L. Finley**, I. Kimber, and D.J. Paustenbach. 2016. A preliminary evaluation of immune stimulation following exposure to metal particles and ions using the mouse popliteal lymph node assay. Tox Appl Pharm. Advance online publication, July 28, 2016. doi: 10.1016/j.taap.2016.07.020.

16. Pierce, J.S., A. Abelmann, J.T. Lotter, P.S. Ruestow, K.M. Unice, E.M. Beckett, H.A. Fritz, J.L. Bare, and **B.L. Finley**. 2016. An assessment of formaldehyde emissions from laminate flooring manufactured in China. Reg Tox Pharm. Advance online publication, July 1, 2016. doi: 10.106/j.yrtph.2016.06.022.

17. Pierce, J.S., A. Abelmann, and **B.L. Finley**. 2016. Comment on "Flavoring Chemicals in E-Cigarettes: Diacetyl, 2,-3-Pentanedione, and Acetoin in a Sample of 51 Products, Including Fruit-, Candy-, and Cocktail-Flavored E-Cigarettes." Environ Health Perspect. 124(6):A100-A101.

18. Cowan, D.M., J.R. Maskrey, E.S. Fung, T.A. Woods, L.M. Stabryla, P.K. Scott, and **B.L. Finley**. 2016. Best-practices approach to determination of blood alcohol concentration (BAC) at specific time points: Combination of ante-mortem alcohol pharmacokinetic modeling and post-mortem alcohol generation and transport considerations. Reg Tox Pharmacol. 78:24-36.

19. Pierce, J.S., P.S. Ruestow, and **B.L. Finley**. 2016. An updated evaluation of reported no-observed adverse effect levels for chrysotile asbestos for lung cancer and mesothelioma. Crit Rev Toxicol. Advance online publication, March 30, 2016. doi: 10.3109/10408444.2016.1150960.

20. Ruestow, P.S., T.J. Duke, **B.L. Finley**, and J.S. Pierce. 2015. Effects of the NFL's amendments to the Free Kick rule on injuries during the 2010 and 2011 season. J Occup Env Hyg. 12(12):875-82.

21. Gaffney, S.H., A. Abelmann, J.S. Pierce, M.E. Glynn, J.L. Henshaw, L.A. McCarthy, J.T. Lotter, M. Liong, and **B.L. Finley**. 2015. Naturally occurring diacetyl and 2,3-pentanedione concentrations associated with roasting and grinding unflavored coffee beans in a commercial setting. Tox Reports. 2:1171-1181.

22. Pierce, J.S. A. Abelmann, J.T. Lotter, C. Comerford, K. Keeton, and **B.L. Finley**. 2015. Characterization of naturally occurring airborne diacetyl concentrations associated with the preparation and consumption of unflavored coffee. Advance online publication, August 18, 2015. doi: 10.1016/j.toxrep.2015.08.006

23. Madl, A.K., M. Liong, M. Kovochich, **B.L. Finley**, D.J. Paustenbach, and G. Oberdorster. 2015. Toxicology of wear particles of cobalt-chromium alloy metal-on-metal hip implants Part I: Physiochemical properties in patient and simulator studies. Nanomed: Nanotech Biol Med. 11(5):1201-1215.

24. Madl, A.K., M. Kovochich, M. Liong, **B.L. Finley**, D.J. Paustenbach, and G. Oberdorster. 2015. Toxicology of wear particles of cobalt-chromium alloy metal-on-metal hip implants Part II: Importance of physiochemical properties and dose in animal and in vitro studies as a basis for risk assessment. Nanomed: Nanotech Biol Med. 11(5):1285-1298.

25. Tvermoes, B.E., D.J. Paustenbach, B.D. Kerger, **B.L. Finley**, and K.M. Unice. 2015. Review of cobalt toxicokinetics following oral dosing: Implications for health risk assessments and metal-on-metal hip implant patients. Crit Rev Toxicol. 45(5):367-87.

26. Kerger, B.D., K.A. Thuett, and **B.L. Finley**. 2014. Evaluation of four α -diketones for toll-like receptor-4 (TLR-4) activation in a human transfected cell line. Food Chem Tox. 74:117-119.

27. **Finley, B.L.** and P.K. Scott. 2014. Diacetyl and "popcorn lung" litigation: Assessing the evidence for general causation. ABA Environ Litig Toxic Torts Newsletter. 15(2):7-10.

28. Paustenbach, D., D. Galbraith, and B. Finley. 2014. Letter to the editor: Authors' response to letters to the editor re: Interpreting cobalt blood concentrations in hip implant patients. Clin Toxicol. 52(5):569-570.

29. Christian, W.V., L.D. Oliver, D.J. Paustenbach, M.L. Kreider, and **B.L. Finley**. 2014. Toxicology-based cancer causation analysis of CoCr-containing hip implants: A quantitative assessment of genotoxicity and tumorigenicity studies. J Appl Tox. 34(9):939-967.

30. Monnot, A.D., W.V. Christian, D.J. Paustenbach, and **B.L. Finley**. 2014. Correlation of blood Cr(III) and adverse health effects: Application of PBPK modeling to determine non-toxic blood concentrations. Crit. Rev. Toxicol. 44(7):618-637.

31. Unice, K.M., B.D. Kerger, D.J. Paustenbach, **B.L. Finley**, and B.E. Tvermoes. 2014. Refined biokinetic model for humans exposed to cobalt dietary supplements and other sources of systemic cobalt exposure. Chem-Biol Interact. 216:53-74.

32. Pierce, J.S., A. Abelmann, L.J. Spicer, R.E. Adams, and **B.L. Finley**. 2014. Diacetyl and 2,3-pentanedione exposures associated with cigarette smoking: Implications for risk assessment of food and flavoring workers. Crit Rev Tox. 44(5):420-435.

33. Tvermoes, B.E., K.M. Unice, D.J. Paustenbach, **B.L. Finley**, J.M. Otani, and D.A. Galbraith. 2014. Effects and blood concentrations of cobalt after ingestion of 1 mg/d by human volunteers for 90 d1-3. Am J Clin Nutr. 99(3):632-46.

34. Paustenbach, D.J., D.A. Galbraith, and **B.L. Finley**. 2014. Interpreting cobalt blood concentrations in hip implant patients. Clin Toxicol. 52(2):98-112.

35. Finley, B.L, K.M. Unice, B.D. Kerger, J.M. Otani, D.J. Paustenbach, D.A. Galbraith, and B.E. Tvermoes. 2013. 31-day study of cobalt (II) chloride ingestion in humans: Pharmacokinetics and clinical effects. J Tox Env Health A. 76:1210-1224.

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Book Chapters

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Presentations at Conferences

1. Ierardi, A.M., J.T. Lotter, N. Jacobs, **B.L. Finley**, and J.S. Pierce. 2021. Derivation of a Proposed Asbestiform Tremolite NOAEL for Lung Cancer. Virtual Poster Presentation at the 2021 Society of Toxicology (SOT) Annual Meeting. March 12-26.

2. Killius, A. and **B.L. Finley**. 2021. Contribution of Demolition Activities to Flint, Michigan, Childhood Blood Lead Levels in 2013-2015. Virtual Poster Presentation at the 2021 Society of Toxicology (SOT) Annual Meeting. March 12-26.

3. Mathis, C., E. Beckett, C. Bates, A. Maier and **B.L. Finley**. 2021. Derivation of adverse Deep Lung Effect Thresholds for α-Diketone Compounds. Virtual Poster Presentation at the 2021 Society of Toxicology (SOT) Annual Meeting. March 12-26.

4. Ierardi, A.M., A. Urban, N.F.B. Jacobs, C. McMenamy, **B.L. Finley** and S.H. Gaffney. 2019. Characterization of Airborne Asbestos Exposures from the use of Dental Products: A Comprehensive Review. Poster presentation at The American Industrial Hygiene Conference & Exposition (AIHce), May 20-22, 2019, Minneapolis, MN.

5. Towle, K.M., A.S. Riordan, J.S. Pierce, **B.L. Finley**, and D.M. Hollins. 2019. Exposure to Asbestos and Risk of Ovarian Cancer: A Systematic Literature Review and Meta-analysis. Poster presentation at The American Industrial Hygiene Conference & Exposition (AIHce), May 20-22, 2019, Minneapolis, MN.

6. Burns; A.M., S.M. Benson, E. Best; D.M. Hollins and **B.L. Finley**. 2018. An Ecological Epidemiology Investigation of Talc Consumption in the US and Trends in Female Cancer Rates. Poster Presentation at Society of Toxicology Annual Meeting, March 11-15, 2018, San Antonio, TX.

7. Kovochich, M. E.S. Fung, S.L. Mahoney, K.M. Unice, D. Fung, D.J. Paustenbach and **B.L. Finley**. 2018. Characterizing Cytotoxic and Inflammatory Responses to Metal-On-Metal Wear Debris from Normal versus Edge-Loading Conditions. Poster Presentation at Society of Toxicology Annual Meeting, March 11-15, 2018, San Antonio, TX.

8. Kovochich, M., D. Fung, K.M. Unice, S.L. Mahoney, D.J. Paustenbach and **B.L. Finley**. 2018. Understanding Divergent Outcomes in MoM-THA Patient Populations with Well-Fixed Components: A Critical Appraisal of Patient Management Protocols and Revision Trends (2000-2017). Poster Presentation at Society of Toxicology Annual Meeting, March 11-15, 2018, San Antonio, TX.

9. Shay Hynds, E.C., A.M. Ierardi, A. M. Burns and **B.L. Finley**. 2018. Derivation of a Chronic Oral RfD for Antimony. Poster Presentation at Society of Toxicology Annual Meeting, March 11-15, 2018, San Antonio, TX.

10. Benson, S.M., G.M. Marsh and **B.L. Finley**. 2017. Cosmetic Talc as a Risk Factor for Mesothelioma: A Weight-of-Evidence Evaluation. Abstract #1290. Poster Presentation at Society of Toxicology Annual Meeting. March 12-16, 2017. Baltimore, MD.

11. **Finley, B.L.**, E.D. Donovan, M. Kovochich, D.J. Paustenbach and A.M. Urban. 2017. Perineal Use of Cosmetic Talc as a Risk Factor for Ovarian Cancer: A Weight-of-Evidence Evaluation. Abstract #1288. Poster Presentation at Society of Toxicology Annual Meeting. March 12-16, 2017. Baltimore, MD.

12. **Finley, B.L.**, M.E. Glynn, W.D. Cyrs, E.W. Beckett, S.H. Gaffney, A.D. Monnot, A. Abelmann, A. Parker and M.A. Maier. 2017. Derivation of an Occupational Exposure Limit for Diacetyl: A Preliminary Analysis. Late Breaking Poster Presentation at Society of Toxicology Annual Meeting. March 12-16, 2017. Baltimore, MD.

13. **Finley, B.L**., P.K. Scott, A.M. Burns and G.M. Marsh. 2017. Does PCB Exposure Cause Non-Hodgkins Lymphoma? A Weight of Evidence Evaluation. Late Breaking Poster Presentation at Society of Toxicology Annual Meeting. March 12-16, 2017. Baltimore, MD.

14. Fung, E.S., A.D. Monnot, B.E. Tvermoes, K.M. Unice, M. Kovochich, D.A. Galbraith, **B.L. Finley** and D.J. Paustenbach. 2017. Characteristics of Cobalt Related Cardiomyopathy in Metal Hip Implant Patients. Abstract #2142. Poster Presentation at Society of Toxicology Annual Meeting. March 12-16, 2017. Baltimore, MD.

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16. Williams, B.H., A.D. Monnot, A. Patton and **B.L. Finley**. 2017. Proposed Chronic Oral Reference Doses for Thallium. Late Breaking Poster Presentation at Society of Toxicology Annual Meeting. March 12-16, 2017. Baltimore, MD.

17. Cyrs, W.D., D. Dussex, M. Glynn and **B.L. Finley**. 2016. Benchmark Dose Modeling for Respiratory Irritation from 2,3-Pentanedione Exposure. (Accepted). Podium Presentation at American Industrial Hygiene Conference & Exposition (AIHce) May 21-26, 2016, Baltimore, MD.

18. Pierce, J.S., A. Abelmann, J. Lotter, C.E. Comerford, K. Keeton and **B.L. Finley**. 2016. Characterization of Naturally Occurring Airborne Diacetyl Concentrations Associated with the Preparation and Consumption of Unflavored Coffee. (Accepted). Poster Presentation at American Industrial Hygiene Conference & Exposition (AIHce) May 21-26, 2016, Baltimore, MD.

19. Pierce, J.S., P.S. Ruestow and **B.L. Finley**. 2016. An Updated Evaluation of Reported Chrysotile Asbestos No Observed Adverse Effect Levels (NOAELs) for Lung Cancer and Mesothelioma. (Accepted). Poster Presentation at American Industrial Hygiene Conference & Exposition (AIHce) May 21-26, 2016, Baltimore, MD.

20. Benson, S.M., S. Batdorf, K.M. Hitchcock, D.A. Galbraith and **B.L. Finley**. 2016. Asbestos exposure and pharyngeal and laryngeal cancer risk: A fiber-type specific meta-analysis. Abstract #2974. Poster Presentation at Society of Toxicology Annual Meeting. March 13-17, 2016. New Orleans, Louisiana.

21. Burns, A.M. and **B.L. Finley**. 2016. Potential tremolite exposures associated with talc- containing products. Abstract #2672. Poster Presentation at Society of Toxicology Annual Meeting. March 13-17, 2016. New Orleans, Louisiana.

22. **Finley, B.L.**, M.E. Glynn, A. Abelmann and J.S. Pierce. 2016. Diacetyl exposure and respiratory disease: an evaluation of the current weight of evidence. Abstract #2140. Poster Presentation at Society of Toxicology Annual Meeting. March 13-17, 2016. New Orleans, Louisiana.

23. Glynn, M.E., R.E. Adams, E.M. Beckett, P.K. Scott, J.S. Pierce and **B.L. Finley**. 2016. Human equivalent concentrations for diacetyl based on minimal bronchiolar respiratory effects observed in animals: A refined analysis using current toxicology data and dosimetry models. Abstract #1300. Poster Presentation at Society of Toxicology Annual Meeting. March 13-17, 2016. New Orleans, Louisiana.

24. Pierce, J.S., P.S. Ruestow and **B.L. Finley**. 2016. An updated evaluation of reported chrysotile asbestos no observed adverse effect levels (NOAELs) for lung cancer and mesothelioma. Abstract #2139. Poster Presentation at Society of Toxicology Annual Meeting. March 13-17, 2016. New Orleans, Louisiana.

25. Tvermoes, B.E., K.M. Unice, B. Winans, M. Kovochich, W.V. Christian, E.D. Donovan, **B.L. Finley**, I. Kimber and D.J. Paustenbach. 2016. Evaluation of immune stimulation following exposure to metal particles and ions using the mouse popliteal lymph node assay. Abstract #3896. Poster Presentation at Society of Toxicology Annual Meeting. March 13-17, 2016. New Orleans, Louisiana.

26. Abelmann, A, KM Unice, SH Gaffney, AM Urban, M Liong, L McCarthy, DM Hollins, JL Henshaw and **B.L. Finley**, Airborne Concentrations of Naturally Occurring Diacetyl Associated with Various Coffee Products. Poster presentation at

the American Industrial Hygiene Conference & Expo (AIHce). May 30-June 4, 2015. Salt Lake City, UT.

27. Gaffney, SH, A Abelmann, JS Pierce, ME Glynn, L McCarthy, J Lotter, M Liong, JL Henshaw and **B.L. Finley**. 2015. Potential Exposure to Naturally Occurring Diacetyl, 2,3-Pentanedione and Respirable Dust from Roasting and Grinding Coffee Beans in an Industrial Setting. Podium presentation at the American Industrial Hygiene Conference & Expo (AIHce). May 30-June 4, 2015. Salt Lake City, UT.

28. Ruestow, PS, JS Pierce, TJ Duke, LJ Roberts and **B.L. Finley**. 2015. Evaluation of the Impact of the "Restraining Line Rule" on the Incidence of Total and Head Injuries Occurring During NFL Kickoffs. Poster presentation at the American Industrial Hygiene Conference & Expo (AIHce). May 30-June 4, 2015. Salt Lake City, UT.

29. Spicer, LJ, J Lotter, CE Comerford, A Abelmann, JS Pierce and **B.L. Finley**. 2015. Pilot Study of Exposures to Airborne Concentrations of Naturally Occurring Diacetyl During Coffee Consumption. Poster presentation at the American Industrial Hygiene Conference & Expo (AIHce). May 30-June 4, 2015. Salt Lake City, UT.

30. Beckett EM, ME Glynn, RE Adams, JS Pierce, PK Scott and **B.L. Finley**. 2015. Refined Derivation of a Human Equivalent Concentration for Hyperplasia of Bronchiolar Epithelium Following Airborne Diacetyl Exposure. Poster presentation at the 54th Annual Meeting and Society of Toxicology (SOT) Meeting at the San Diego Convention Center, San Diego, CA; March 22-26, 2015.

31. Christian, WV, LD Oliver, DJ Paustenbach, ML Kreider and **B.L. Finley**. 2015. Toxicology Based Cancer Causation Analysis of CoCr-Containing Hip Implants: A Quantitative Assessment of Genotoxicity and Tumorigenicity Studies. Poster presentation at the 54th Annual Meeting and Society of Toxicology (SOT) Meeting at the San Diego Convention Center, San Diego, CA; March 22-26, 2015.

32. Cowan, DM, JR Maskrey, E Fung, T Woods, L Stabryla, PK Scott and **B.L. Finley**. 2015. Evaluation of Empirically-based PBPK Modeling Approaches for Modeling Ante- and Post Mortem Ethanol Concentrations in Biological Matrices. Poster presentation at the 54th Annual Meeting and Society of Toxicology (SOT) Meeting at the San Diego Convention Center, San Diego, CA; March 22-26, 2015.

33. Glynn, ME, RE Adams, EM Beckett, JS Pierce, PK Scott and **B.L. Finley**. 2015. Derivation of a Human Equivalent Concentration for Chronic Inflammation in the Bronchial and Bronchiolar Epithelium of the Lung Following Inhalation Exposure to Diacetyl. Poster presentation at the 54th Annual Meeting and Society of Toxicology (SOT) Meeting at the San Diego Convention Center, San Diego, CA; March 22-26, 2015.

34. Pierce, JS, A Abelmann, J Lotter, C Comerford, K Keeton and **B.L. Finley**. 2015. Characterization of Diacetyl Exposures Associated with the Preparation and Consumption of Unflavored Coffee. Poster presentation at the 54th Annual Meeting and Society of Toxicology (SOT) Meeting at the San Diego Convention Center, San Diego, CA; March 22-26, 2015.

35. Urban, AM, SH Gaffney, L McCarthy, DM Hollins, A Abelmann, K Unice, M Liong, **B.L. Finley**, JL Henshaw. 2015. Volatile Organic Compounds Released from Spreading Ground Coffee during a Simulated Industrial Task. Poster presentation at the 54th Annual Meeting and Society of Toxicology (SOT) Meeting at the San Diego Convention Center, San Diego, CA; March 22-26, 2015.

36. Christian WV, LD Oliver, ML Kreider ML and **B.L. Finley**. 2014. Toxicology-based cancer causation analysis of CoCr-containing hip implants: A quantitative assessment of in vitro genotoxicity studies. Poster presentation at the 2014 Society for Risk Analysis Annual Meeting, December 7 - 10, 2014. #P133, Plaza Ballroom ABC, 6:00 to 8:00 pm, December 8, 2014. Denver, CO.

37. Pierce, J.S., A. Abelmann, L.J. Spicer, R.E. Adams, **and B.L. Finley**. Diacetyl and 2,3-Pentanedione Exposures Associated with Cigarette Smoking: Implications for Risk Assessment of Food and Flavoring Workers. Presented at the American Industrial Hygiene Conference & Exposition (AIHce); San Antonio, TX; May 31-June 5, 2014.

38. Adams, R.E., M.E. Glynn, J.S. Pierce, and **B.L. Finley**. 2014. Derivation of A Human Equivalent Concentration for Diacetyl for Hyperplasia of the Bronchiolar Epithelium. Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo; Phoenix, AZ; March 23-27, 2014.

39. Christian, W.V., L.D. Oliver, M.L. Kreider, and **B.L. Finley**. 2014. In vitro Genotoxicity Assays with Co and Cr(III) Ions and Alloy Particles: Implications for Cancer Risks to Hip Implant Patients. Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo; Phoenix, AZ; March 23-27, 2014.

40. **Finley, B.L.**, J.S. Pierce, K. Neier and L.J. Roberts. 2014. Weight-of-Evidence Evaluation of the Respiratory Effects Associated with Diacetyl. Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo; Phoenix, AZ; March 23-27, 2014.

41. Fritz, H.A., M.E. Glynn, K.E. Neier, J.S. Pierce, A.M. Urban, S.H. Gaffney, and **B.L. Finley**. 2014. Comparison of the Upper Respiratory Tract Uptake Efficiencies of Vapors in Rats and Mice. Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo; Phoenix, AZ; March 23-27, 2014.

42. Gaffney, S.H, C.J. Ronk, **B.L. Finley**, J.L. Henshaw, J.S. Pierce, L. McCarthy, J. Lotter, and A. Abelmann. 2014. Naturally-Occurring Airborne Diacetyl

Concentrations Resulting from Roasting and Grinding Coffee Beans in an Industrial Setting. Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo; Phoenix, AZ; March 23-27, 2014.

43. Grespin, M.E., E.D. Donovan, R.J. Ward, A.K. Madl, and **B.L. Finley**. 2014. Asbestos Content of Heavy Equipment Brake-Wear Debris and Associated Airborne Exposure During Brake Work. Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo; Phoenix, AZ; March 23-27, 2014.

44. Kerger, B.D, C.J. Ronk, M.E. Glynn, **B.L. Finley**, and D.J. Paustenbach. 2014. Age-Related Trends in US Pleural Mesothelioma and Soft Tissue Sarcoma Rates: Evidence for a Longevity Effect. Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo; Phoenix, AZ; March 23-27, 2014.

45. Liong, M., M. Kovochich, **B.L. Finley**, D.J. Paustenbach, and A.K. Madl. 2014. Nanoparticles from the Wear of Cobalt-Chromium Alloy Metal-on-Metal Hip Implants: Physicochemical and Dose Analysis of Patient and Toxicology Studies. Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo; Phoenix, AZ; March 23-27, 2014.

46. Monnot, A.D., W.V. Christian, **B.L. Finley**, and D.J. Paustenbach. 2014. Correlation of Blood Cr (III) and Adverse Health Effects: Application of PBPK Modeling to Determine Non-toxic Blood Concentrations. Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo; Phoenix, AZ; March 23-27, 2014.

47. Neier, K., M.E. Glynn, H. Fritz, A. Urban, S.H. Gaffney, and **B.L. Finley**. 2014. Quantitative Evaluation Of The Relationship Between Vapor Characteristics And Upper Respiratory Tract Uptake Efficiencies In Rodents Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo; Phoenix, AZ; March 23-27, 2014.

48. Pierce, J.S., A. Abelmann, L.J. Spicer, R.E. Adams, and **B.L. Finley**. 2014. Concentrations of Diacetyl and 2,3-Pentanedione in Mainstream Cigarette Smoke: A Comparison to Workplace Exposures. Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo; Phoenix, AZ; March 23-27, 2014.

49. Thuett, K.A., B.D. Kerger, **B.L. Finley**, and D.J. Paustenbach. 2014. Evaluation of Four Alpha-Diketones for Toll-Like Receptor-4 (TLR-4) Activation in Human Embryonic Kidney Cells. Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo; Phoenix, AZ; March 23-27, 2014.

50. Glynn, M.E., J.S. Pierce, B.H. Williams, L.E. Johns, R. Adhikari and **B.L. Finley**. 2013. Residential and occupational exposure to wood treating operations and bladder cancer: A meta-analysis. Poster presentation at the 2013 Society for Risk Analysis Annual Meeting, December 8 - 11, 2013. #P36, Key Ballroom, December 9, 2013. Baltimore, MD.

51. Williams, B.H., J.S. Pierce, M.E. Glynn, L.E. Johns, R. Adhikari and **B.L. Finley**. Residential and Occupational Exposure to Wood Treating Operations and Risk of Non-Hodgkin Lymphoma: A Meta-Analysis. Poster presentation at the 2013 Society for Risk Analysis Annual Meeting, December 8 - 11, 2013. #P35, Key Ballroom, December 9, 2013. Baltimore, MD.

52. Monnot, A., A.L. Perez and **B.L. Finley**. 2013. Establishing Blood Biological Screening Indices for Cobalt Exposure. Invited platform presentation: Society of Environmental Toxicology and Chemistry, 34th Annual Meeting, Nashville, TN, November 11-14, 2013.

53. Johns, L. E., J. S. Pierce, A. Abelmann, and **B.L. Finley**. Residential and Occupational Exposure to Wood Treating Operations as Risk Factors for Bladder Cancer. Society for Epidemiologic Research 46th Annual Meeting. June 18-21, 2013. Boston, MA.

54. Pierce J.S, A. Abelmann, L.J. Spicer, L.E. Johns, L.J. Roberts, H.A. Fritz, M.E. Glynn, and **B.L. Finley**. 2013. A Comparison of Reported Peak Concentrations of Diacetyl in the Workplace and Estimated Diacetyl Concentrations in Mainstream Cigarette Smoke. Session: Indoor Environmental Quality. Poster presentation at The American Industrial Hygiene Conference & Exposition (AIHce) in Montreal, Quebec, Canada. May 20, 2013; 10:00 AM - 12:00 PM. Presentation Number: SR-401-11

55. Spicer, L.J., J.S. Pierce, M.E. Glynn, A. Abelmann, L.J. Roberts, L.E. Johns, H.A. Fritz, and **B.L. Finley** . 2013. A Comparison of Workday-Duration Occupational Exposures to Diacetyl to Estimated Exposures Associated with Cigarette Smoking. Session: IH General Practice. Poster presentation at The American Industrial Hygiene Conference & Exposition (AIHce) in Montreal, Quebec, Canada. May 20, 2013; 2:00 PM - 4:00 PM. Presentation Number: SR-402-03

56. Vishnevskaya, L., K.A. Thuett, W.D. Cyrs, P.S. Chapman, D.J. Paustenbach, and **B.L. Finley**. 2013. Evolution of Warnings and Labels on Encapsulated Asbestos-Containing Products (1930?1990). Session: Legal, Regulatory, Guidelines, Standards. Poster presentation at The American Industrial Hygiene Conference & Exposition (AIHce) in Montreal, Quebec, Canada. May 21, 2013; 10:00 AM - 12:00 PM. Presentation Number: SR-403-08

57. **Finley, B.L.**, B.E. Tvermoes, K.M. Unice, J.M. Otani, D.J. Paustenbach and D.A. Galbraith. 2013. Cobalt whole blood concentrations in healthy adult volunteers following two-weeks of ingesting a cobalt supplement. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013 at the Henry B. Gonzalez Convention Center in San Antonio, Texas. March 12, 2013.

58. Kerger, B.D., R. Gerads, **B.L. Finley** and D.J. Paustenbach. 2013. Method: measuring protein-bound and free cobalt(II) in human serum - size exclusion liquid

chromatography with ICP-MS. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013 at the Henry B. Gonzalez Convention Center in San Antonio, Texas.

59. Monnot, A.D., S.H. Gaffney, D.J. Paustenbach, and **B.L. Finley**. 2013. Dose-response relationships for blood cobalt concentrations and associated health effects. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013 at the Henry B. Gonzalez Convention Center in San Antonio, Texas.

60. Paustenbach, D.J., B.E. Tvermoes, J.M. Otani, K.M. Unice, **B.L. Finley**, and D.A. Galbraith. 2013. Cobalt blood concentrations and health effects in adult volunteers during a 90 day cobalt supplement ingestion study. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013 at the Henry B. Gonzalez Convention Center in San Antonio, Texas.

61. Thuett, K.A., **B.L. Finley**, P.K. Scott and D.J. Paustenbach. 2013. Speciation of Chromium Released from Metal-on-Metal Hip Implants. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013 at the Henry B. Gonzalez Convention Center in San Antonio, Texas.

62. Tvermoes, B.E., **B.L. Finley**, J.M. Otani, K.M. Unice, D.J. Paustenbach and D.A. Galbraith. 2013. Effects of cobalt dietary supplementation on cobalt body burden, steady-state levels and selected biochemical parameters. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013 at the Henry B. Gonzalez Convention Center in San Antonio, Texas.

63. **Finley, B.L.**, J.S. Pierce, A. Abelmann, L.J. Spicer, R.E. Adams. 2012. Diacetyl Eexposure from cigarette smoke: Implications for assessing diacetyl exposure-response in popcorn and flavor manufacturing workers. Presented at the 2012 Society for Risk Analysis (SRA) Annual Meeting; December 9-12, 2012; San Francisco, CA.

64. Monnot, A.D., Gaffney, S.H., Paustenbach, D.J., and **Finley, B.L.** Derivation of a Chronic Oral Reference Dose for Cobalt. Presented at the 2012 Society for Risk Analysis Annual Meeting; December 9-12, 2012; San Francisco, CA.

65. Roberts, L.J., J.S. Pierce, L.E. Johns, L. Lievense, and **B.L. Finley**. 2012. Weight of evidence evaluation of diacetyl dose response relationship in popcorn and flavoring worker studies. Presented at the 2012 Society for Risk Analysis (SRA) Annual Meeting; December 9-12, 2012; San Francisco, CA.

66. Tvermoes, B.E., J. Otani, K. Unice, **B. Finley**, D.J. Paustenbach, D. Galbraith. Investigation of Cobalt Steady-State Levels in Five Healthy Adult Volunteers Taking 14-Days of a Cobalt Supplement. Presented at the 2012 Society for Risk Analysis Annual Meeting; December 9-12, 2012; San Francisco, CA.

67. Abelmann A., J.S. Pierce, L.J. Spicer, R.E. Adams, and **B.L. Finley**. 2012. Exposure to diacetyl vapors associated with secondhand cigarette smoke. Presented at the 2012 Annual Meeting of The International Society of Exposure Science (ISES). October 28 - November 1, in Seattle, WA.

68. Pierce, J.S., A. Abelmann, L.J. Spicer, R.E. Adams, and **B.L. Finley**. 2012. Diacetyl exposure from active cigarette smoking: implications for characterizing health risks in popcorn and flavor manufacturing workers. Presented at the 2012 Annual Meeting of The International Society of Exposure Science (ISES). October 28 - November 1, in Seattle, WA.

69. Johns, L.E., J.S. Pierce, L.J. Roberts, L.J. Lievense and **B.L. Finley**. 2012. Assessment of the exposure-response relationship between diacetyl and respiratory health outcomes among popcorn and flavor manufacturing workers. Presented at the 45th Society for Epidemiologica Research (SER) Annual Meeting. June 27 - 30, in Minneapolis, MN.

70. Pierce, J.S., L.J. Spicer, A. Abelmann, R.E. Adams, L. Roberts, L. Johns, L., D.M. Hollins and **B.L. Finley**. 2012. Characterization of airborne diacetyl concentrations associated with consuming different types of wine. Presented at the Institute of Food Technologists (IFT) 2012 Annual Meeting & Food Expo June 25 - June 28, in Las Vegas Nevada.

71. Pierce, J.S., A. Abelmann, L.J. Spicer, R.E. Adams, **B.L. Finley** and S.H. Gaffney. 2012. Characterization of formaldehyde exposure resulting from the use of four professional hair straightening products. Presented at 2012 American Industrial Hygiene Conference & Expo (AIHce), June 16-21, in Indianapolis, IN.

72. Spicer, L.J., J.S. Pierce, A. Abelmann, R.E. Adams, **B.L. Finley** and S.H. Gaffney. 2012. Analysis of time weighted average concentrations of formaldehyde during keratin hair smoothing treatments. Presented at 2012 American Industrial Hygiene Conference & Expo (AIHce), June 16-21, in Indianapolis, IN.

73. Kreider, M.L., J.M. Panko and **B.L. Finley**. 2012. Effects of subacute inhalation exposure to tire and road wear particles in rats. Presented at the 2012 Health Effects Institute (HEI) Annual Conference. Chicago, IL, April 15-17, 2012.

74. Adams R., **B.L. Finley**, J.S. Pierce, A.D. Phelka, D.J. Paustenbach, K. Thuett and C. Barlow. 2012. Derivation of LOAEL and NOAEL for tremolite asbestos. Presented at Society of Toxicology's (SOT) 51st Annual Meeting, March 11-15, 2012, at the Moscone Convention Center in San Francisco, California. March 14, 2012. Exposure Assessment: Case-Specific Characterizations; 9:00 AM - 12:30 PM; Exhibit Hall; 2114 Poster Board -538.

75. Knutsen J.S., B.D. Kerger, **B.L. Finley** and D.J. Paustenbach. 2012. Human PBPK modeling of benzene inhalation based Chinese worker urinary metabolite data:

comparison of human and mouse metabolism. Presented at Society of Toxicology's (SOT) 51st Annual Meeting, March 11-15, 2012, at the Moscone Convention Center in San Francisco, California. March 14, 2012. Disposition and Pharmacokinetics; 9:00 AM - 12:30 PM; Exhibit Hall; 1893 Poster Board -226.

76. Phelka A., **B.L. Finley**, J. Pierce, R. Adams, D.J. Paustenbach, K. Thuett and C. Barlow. 2012. Tremolite asbestos exposures associated with the use of commercial products. Presented at Society of Toxicology's (SOT) 51st Annual Meeting, March 11-15, 2012, at the Moscone Convention Center in San Francisco, California. March 14, 2012. Exposure Assessment: Case-Specific Characterizations; 9:00 AM - 12:30 PM; Exhibit Hall; 2115 Poster Board -539.

77. **Finley, B.L.**, S. Gaffney, A. Abelmann, and J.S. Pierce. Evaluation of Pentachlorophenol and PCDD/Fs Exposure as Risk Factors for Multiple Myeloma. International Symposium on Halogenated Persistent Organic Pollutants - Dioxin 2011. August 21-25, 2011. Brussels, Belgium.

78. Hollins, D., D. Galbraith, **B. Finley**, and J. Pierce. Occupational Exposure to Artificial Butter Flavorings: A State-of-the-Art Analysis. Presentation at American Industrial Hygiene Conference and Expo (AIHce), May 14 - May 19, 2011, Portland, Oregon. PO 121-2. Wednesday May 18, 2011. 10:20am. OCC Oregon 204.

79. Kreider, M., J. Panko, J. McDonald, B. McAtee, **B.L. Finley** and J. Seagrave. 2011. Effects of intratracheal instillation of tire and road wear particles (TRWP) and tread particles (TP) on inflammation and cytotoxicity in rat lung: A comparative toxicity study. Presented at the Society of Toxicology (SOT) Annual Meeting, Wednesday March 9, 2011.

80. **Finley, B.L.**, L.L.F. Scott and D.A. Galbraith. 2010. Pleural mesothelioma in U.S. auto mechanics: Expected vs. reported cases from 1975-2007. Presented at the 2010 Society for Risk Analysis Conference. December 5-8, 2010. Salt Lake City, UT.

81. Phelka, A.D. and **B.L. Finley**. 2010. Potential health hazards associated with exposures to asbestos-containing drywall accessory products: A state-of-the-science assessment. Presented at the 2010 Society for Risk Analysis Conference. December 5-8, 2010. Salt Lake City, UT.

82. Hollins, D.M., D.A. Galbraith and **B.L. Finley**. 2010. Occupational exposure to diacetyl and potential health effects: A weight of evidence analysis. Poster presentation at the 2010 Society for Risk Analysis Conference. Poster #47. December 5-8, 2010. Salt Lake City, UT.

83. Shay, E., K. Thuett and **B.L. Finley**. 2010. Atrazine in Drinking water: Comparison of measured and estimated peak concentration vs. acute health benchmarks. Poster presentation at the 2010 Society for Risk Analysis Conference. Poster #49. December 5-8, 2010. Salt Lake City, UT.

84. Marwood C., B.L. McAtee, M.L. Kreider, R. Ogle, **B.L. Finley** and J.M. Panko.2010. Identification of toxic constituents in tire and road wear particle extracts. Poster presentation at the 2010 Society of Environmental Toxicology and Chemistry (SETAC) Conference. November 7-11, 2010. Portland, OR.

85. Marwood C, B.L. McAtee, M.L. Kreider, **B.L. Finley**, J.M. Panko. 2010. Chronic toxicity of tire/road wear particles in sediment to aquatic organisms. Poster presentation at the 2010 Society of Environmental Toxicology and Chemistry (SETAC) Conference. November 7-11, 2010. Portland, OR.

86. Fillos, D.J., W.J. Luksemburg, M. Anderle de Sylor, L.L.F. Scott and **B.L. Finley**. 2010. Measurements of PCDD and PCDF concentrations in wild-caught and farm-raised shrimp from the U.S. retail market. Poster presentation at the 30th International Symposium on Halogenated Persistent Organic Pollutants. Abstract #1498, Poster Board #59. September 12-17, 2010. San Antonio, TX.

87. Fillos, D.J., W.J. Luksemburg and **B.L. Finley**. 2010. TEQ calculations and daily intake estimates associated with PCBs in shrimp from the U.S. retail market. Poster presentation at the 30th International Symposium on Halogenated Persistent Organic Pollutants. Abstract #1624, Poster Board #609. September 12-17, 2010. San Antonio, TX.

88. Scott, P.K. Fillos, D., W.J. Luksemburg and **B.L. Finley**. 2010. Principal components analysis (PCA) of PCB concentrations in shrimp from the U.S. retail market. Poster presentation at the 30th International Symposium on Halogenated Persistent Organic Pollutants. Abstract #1497, Poster Board #259. September 12-17, 2010. San Antonio, TX.

89. Scott, L.F., D.A. Galbraith and **B.L. Finley**. 2010. Mesothelioma in U.S. automotive mechanics: Reported vs expected number of cases in 1975-2007. Poster presentation at the Society of Toxicology's Annual Meeting. #2312PB643. March 7-11, 2010. Salt Lake City, UT.

90. Donovan, B., E.P. Donovan, S.H. Gaffney, P. Scott and **B.L. Finley**. 2010. Human health risks associated with fish and shellfish consumption in an industrial leasehold in a southern California bay. Poster presentation at the Society of Toxicology's Annual Meeting #2315PB646. March 7-11, 2010. Salt Lake City, UT.

91. McAtee, B.L., Kreider, M.L., Panko, J.M., Sweet, L.I., **Finley, B.L.** Biological leaching of metals from respirable tire wear particles. Presentation at 46th Congress of the European Societies of Toxicology. September 13-16, 2009. Dresden, Germany.

92. Panko, J.M., McAtee, B.L., Kreider, M.L., Gustafsson, M., Blomqvist, G., Gudmundsson, A., Sweet, L.I., **Finley, B.L.** Physio-Chemical Analysis of Airborne

Tire Wear Particles. Presentation at 46th Congress of the European Societies of Toxicology. September 13-16, 2009. Dresden, Germany.

93. **Finley, B.L.** The Use of Risk Assessment Principles in Welding Rod Litigation. Presenter at Global Welding Practices: Manganese Exposure Assessment, Control, and Litigation (RT 241) at American Industrial Hygiene Conference and Expo (AIHce), May 31 - Jun 4, 2009, Toronto, Canada

94. Scott, L.F., J. Keenan, **B.L. Finley** and S.H Gaffney. 2009. Using blood measurements to assess exposure to dioxin/furans: Potential influence of elevated detection limits. Presented at the Society of Toxicology's Annual Meeting. #1621 PB 533. March 15-19, 2009. Baltimore, MD.

95. Williams, C., E.C. Shay, K.M. Unice, P.K. Scott and **B.L. Finley**. 2009. Evaluation of fluoropolymer toxicity from outbreak reports and animal studies. Poster presentation at The Society of Toxicology's Annual Meeting. #1208 PB 533. March 15-19, 2009. Baltimore, MD.

96. Donovan, E.P., B.L. Donovan, S.H. Gaffney, P.K. Scott and **B.L. Finley**. 2008. Human health risks associated with fish and shellfish consumption in an industrialized leasehold in a southern California bay. Abstract #1723. Presented at the International Society for Environmental Epidemiology & International Exposure Analysis (ISEE/ISEA) Joint Annual Conference, October 12-16, 2008. Pasadena, CA.

97. **Finley, B.L.** 2008. A screening level evaluation of noncancer hazards for occupational exposures to decachlorobiphenyl from indoor dust. Poster presentation at the 28th International Symposium on Halogenated Persistent Organic Pollutants (POPs) - Dioxin Conference. #321. August 17-22, 2008. Birmingham, England, U.K.

98. **Finley, B.L.** 2008. Site-specific dermal risk assessment for industrial workers. Poster presentation at the 28th International Symposium on Halogenated Persistent Organic Pollutants (POPs) - Dioxin Conference. #305. August 17-22, 2008. Birmingham, England, U.K.

99. **Finley, B.L.**, A.K. Madl, D.M. Murbach, K.A. Fehling, D.J. Paustenbach and G.C. Jiang. 2008. A study of airborne chrysotile concentrations associated with handling, unpacking and repacking boxes of automobile clutch discs (circa 1950-1980). Abstract # 1458. Presented at Society of Toxicology's 47th Annual Meeting, March 16-20, 2008. Seattle, WA.

100. Haws, L.C., M.J. DeVito, J.N. Walker, L.S. Birnbaum, K.M. Unice, P. Scott, M.A. Harris, J.A. Tachovsky, W.H. Farland, **B.L. Finley** and D.F. Staskal. 2008. Development of weighted distributions of REPs for dioxin-like compounds:

implications for risk assessment. Abstract # 1178. Presented at Society of Toxicology's 47th Annual Meeting, March 16-20, 2008. Seattle, WA.

101. Haws, L.C., M.J. DeVito, J.N. Walker, L.S. Birnbaum, K.M. Unice, P. Scott, M.A. Harris, J.A. Tachovsky, W.H. Farland, B. Finley, and D.F. Staskal. Development of Weighted Distributions of REPs for Dioxin-Like Compounds: Implications for Risk Assessment. Poster presentation at the Society of Toxicology's Annual Meeting. #617. March 16-20, 2008. Seattle, WA.

102. Jiang, G.C., A.K. Madl, D.J. Paustenbach and **B.L. Finley**. 2008. Detailed asbestos fiber size and morphology analyses of automobile clutch discs, brake pads and brake shoes. Abstract # 1468. Presented at Society of Toxicology's 47th Annual Meeting, March 16-20, 2008. Seattle, WA.

103. Phelka, A.D., J.A. Clarke, D.J. Paustenbach and **B.L. Finley**. 2008. The importance of asbestos fiber length as a predictor of potency for asbestos-related disease. Abstract # 1185. Presented at Society of Toxicology's 47th Annual Meeting, March 16-20, 2008. Seattle, WA.

104. Scott, L., L. Haws, D.F. Staskal, M. Harris and **B.L. Finley**. 2008. Evaluation of dioxin-like compounds in workers from a primary magnesium production facility relative to levels observed in the general US population. Abstract # 1741. Presented at Society of Toxicology's 47th Annual Meeting, March 16-20, 2008. Seattle, WA.

105. Staskal, D.E., E.P. Donovan, K.M. Unice, L.C. Haws, J. Roberts, **B.L. Finley** and M. Harris. 2008. Human health risks associated with exposure to pathogens in waters and sediments of the lower Passaic River. Abstract # 1800. Presented at Society of Toxicology's 47th Annual Meeting, March 16-20, 2008. Seattle, WA.

106. Jiang G.C-T., A.K. Madl, D.M. Murbach, K.A. Fehling, **B.L. Finley**. Exposure to Chrysotile Asbestos Associated with Handling, Unpacking, and Repacking Boxes of Automobile Clutch Discs. 17th Annual Conference of the International Society for Exposure Analysis. October 14-18, 2007. Raleigh, NC.

107. **Finley, B.L.**, E.J. Morinello and K.A. Fehling. 2007. Relative oral bioavailability of polychlorinated dibenzo-p-dioxins / dibenzofurans in soil. Abstract # P258. Presented at Dioxin 2007. September 2-7, 2007. Tokyo, Japan.

108. Haws, L.C., L.L.F. Scott, D.F. Staskal, M.A. Harris and **B.L. Finley**. 2007. Dioxin-like compounds in workers at a primary magnesium production facility. Abstract # P261. Presented at Dioxin 2007. September 2-7, 2007. Tokyo, Japan.

109. **Finley, B.L.** and J. Pierce. 2007. Characterization of risks to welders due to exposures to asbestos in welding rod flux. Abstract #1922-211. Presented at Society of Toxicology's 46th Annual Meeting, March 25-29, 2007. Charlotte, NC.

110. Haws, L., N. Walker, M. DeVito, L. Birnbaum, K. Unice, P. Scott, M. Harris, W. Farland, **B.L. Finley** and D. Staskal. 2007. Development of weighted distributions of reps for dioxin-like compounds (DLCs). Abstract # 1560. Presented at Society of Toxicology's 46th Annual Meeting, March 25-29, 2007. Charlotte, NC.

111. Morinello, E.J., J.M. Warmerdam and **B.L. Finley**. 2007. Absolute oral bioavailability of polychlorinated dibenzo-p-dioxins/dibenzofurans in soil. Abstract # 825-302. Presented at Society of Toxicology's 46th Annual Meeting. March 25-29, 2007. Charlotte, NC.

112. Moy, P., J. Pierce and **B.L. Finley**. 2007. Assessment of health effects resulting from possible emissions from a pesticide formulation facility. Abstract # 1540-415. Presented at Society of Toxicology's 46th Annual Meeting. March 25-29, 2007. Charlotte, NC.

113. Pierce, J., J. Warmerdam and **B.L. Finley**. 2007. Estimating health risks in workers aboard crude oil tankers due to exposure to n-hexane, toluene and benzene. Abstract # 1999-218. Presented at Society of Toxicology's 46th Annual Meeting. March 25-29, 2007. Charlotte, NC.

114. Roberts, J., D.A. Galbraith and **B.L. Finley**. 2007. A comprehensive review of occupational exposure to diacetyl in microwave popcorn facilities. Abstract # 2000-219. Presented at Society of Toxicology's 46th Annual Meeting. March 25-29, 2007. Charlotte, NC.

115. Staskal, D., E. Donovan, J. Roberts, K. Unice, **B.L. Finley** and M. Harris. 2007. Human health risk associated with exposure to pathogen-contaminated sediments. Abstract # 1618-120. Presented at Society of Toxicology's 46th Annual Meeting. March 25-29, 2007. Charlotte, NC.

116. Warmerdam, J.M., E.J. Morinello and **B.L. Finley**. 2007. Relative oral bioavailability of polychlorinated dibenzo-p-dioxins/dibenzofurans in soil. Abstract # 824-301. Presented at Society of Toxicology's 46th Annual Meeting. March 25-29, 2007. Charlotte, NC.

117. Paustenbach, D.J., J.R. Kuykendall, **B.L. Finley**, J.M. Warmerdam, and P.P. Moy. Presented by J.S. Knutsen. Factors Affecting Bioaccessibility and Bioavailability of Hexavalent Chromium and Dioxin Contaminants in Soil, and Their Relevance to Risk Assessment. International Conference on Environmental Epidemiology and Exposure. September 2-6, 2006. #MS1-05. Paris, France.

118. Haws, L.C., M.J. DeVito, L.S. Birnbaum, N.J. Walker, P.K. Scott, P.K., K.M. Unice, M.A. Harris, W.H. Farland, **B.L. Finley**, and D.F. Staskal. An alternative method for establishing TEFs for dioxin-like compounds. Part 2. Development of an approach to quantitatively weight the underlying potency data. 26th International Symposium on

Halogenated Persistent Organic Pollutants-Dioxin. August 21-25, 2006. Oslo, Norway.

119. Scott, P.K., L.C. Haws, D.F. Staskal, L.S. Birnbaum, N.J. Walker, M.J. DeVito, M.A. Harris, W.H. Farland, **B.L. Finley**, and K.M. Unice. An alternative method for establishing TEFs for dioxin-like compounds. Part 1. Evaluation of decision analysis methods for use in weighting relative potency data. 26th International Symposium on Halogenated Environmental Organic Pollutants and POPs-Dioxin. August 21-25, 2006. Oslo, Norway.

120. Staskal, D.F., K. Unice, N.J. Walker, M.J. DeVito, L.S. Birnbaum, P.K. Scott, M.A. Harris, W.H. Farland, **B.L. Finley**, and L.C. Haws. Part 3. An Alternative Method for Establishing TEFs for Dioxin-like Compounds. Development of Weighted Distributions of REPs for PCB126 and 2,3,4,7,8-PeCDF. International Symposium on Halogenated Persistent Organic Pollutants and POPs-Dioxin. August 21-25, 2006. # 68. Oslo, Norway.

121. Hong, S., D. Proctor and **B.L. Finley**. 2006. Assessment of L.A. sewage spills on Santa Monica Bay beaches. Abstract #1612. Presented at Society of Toxicology's 45th Annual Meeting. March 5-9, 2006. San Diego, CA.

122. Mellinger, K.N., J.R. Kuykendall, K.L. Miller, A.V. Cain, **B.L. Finley** and D.J. Paustenbach. 2006. DNA-Protein crosslinks as a potential biomonitor of hexavalent chromium exposure in rainbow trout. Abstract # 1264. Presented at Society of Toxicolog's 45th Annual Meeting. March 5-9, 2006. San Diego, CA.

123. Perry, M.W., J.R. Kuykendall, K.L. Miller, K.N. Mellinger, A.V. Cain, **B.L. Finley** and D.J. Paustenbach. 2006. Persistence of DNA-Protein crosslinks in erythrocytes of channel catfish after acute hexavalent chromium exposure. Abstract # 1266. Presented at Society of Toxicology's 45th Annual Meeting. March 5-9, 2006. San Diego, CA.

124. Pierce, J.S. and **B.L. Finley**. 2006. Characterization of chrysotile asbestos exposures for garage mechanics. Abstract #:854. Presented at Society of Toxicology's 45th Annual Meeting. March 5-9, 2006. San Diego, CA.

125. Scott, P., M.A. Harris, **B.L. Finley**, L. Ferriby, and R. Budinsky. Development of a Relative Estimate of Potency Distribution for 2,3,7,8-TCDF: A Proposed Approach. 25th International Symposium on Halogenated Environmental Organic Pollutants and POPs-Dioxin. August 21-26, 2005. Toronto, Canada.

126. **Finley, B.L.**, F. Mowat, R. Richter, G. Brobry, V. Craven and P. Sheehan. 2005. Evaluation of Proposed Threshold doses for chrysotile exposure and respiratory diseases. Abstract # 409. Presented at Society of Toxicology's 44th Annual Meeting. March 6-10. New Orleans, LA.

127. Harris, M.A. and **B.L. Finley**. 2005. The TCDD TEQ in human blood from dietary vs. anthropogenic dioxins: A dietary study. Abstract #392. Presented at Society of Toxicology's 44th Annual Meeting. March 6-10. New Orleans, LA.

128. **Finley, B.L.**, R.O. Richter, F.S. Mowat, S. Mlynarek, D.J. Paustenbach, J.L. Warmerdam, and P.J. Sheehan. Cumulative Occupational Asbestos Exposures of U.S. Brake Repair Mechanics. Annual Meeting of the Society for Risk Analysis Conference. December 5-8, 2004. #T6.2. Palm Springs, CA.

129. Paustenbach, D.J., R.O. Richter, **B.L. Finley**, P. Williams, and P.J. Sheehan. Evaluating Asbestos Exposures Associated with Vehicle Brake Cleaning and Machining Activities using Short-Term and TWA Measurements. Annual Meeting of the Society for Risk Analysis. December 5-8, 2004. #T6.3. Palm Springs, CA.

130. Richter, R.O., **B.L. Finley**, P.J., D.J. Paustenbach, and P. Williams. Short-Term Asbestos Exposures Associated with Vehicle Brake Cleaning and Machining Activities from 1970 to 1990. International Society of Exposure Analysis' Annual Conference. October 17-21, 2004. #W2D-01. Philadelphia, PA.

131. Connor, K., M.A. Harris, M.A. Edwards, A. Chu, G. Clark, and **B.L. Finley**. Estimating the total TEQ in human blood from naturally-occurring vs.. anthropogenic dioxins: a dietary study. 24th International Symposium of Halogenated Environmental Organic Pollutants and POPs-Dioxin. September 6-10, 2004. Berlin, Germany.

132. Haws, L.C, M.A. Harris, S. Su, N. Walker, L. Birnbaum, M.J. DeVito, W. Farland, K.Connor, A. Santamaria, and **B.L. Finley**. A preliminary approach to characterizing variability and uncertainty in the mammalian PCDD/F and PCB TEFs. International Symposium of Halogenated Environmental Organic Pollutants and POPs-Dioxin. September 6-10, 2004. Berlin, Germany.

133. Haws, L.C., M.A. Harris, S. Su, L.Birnbaum, M.J. DeVito, W. Farland, N. Walker, K.Connor, A. Santamaria, and **B.L. Finley**. Development of a refined database of relative potency estimates to facilitate better characterization of variability and uncertainty in the current mammalian TEFs for PCDDs, PCDFs, and dioxin-like PCBs. International Symposium of Halogenated Environmental Organic Pollutants and POPs-Dioxin. September 6-10, 2004. Berlin, Germany.

134. Li, A.A., P. Mink, L. McIntosh, J. Teta and **B.L. Finley**. 2004. Evaluation of epidemiological and animal data associating pesticides with Parkinson's Disease. Abstract #1352. Presented at Society of Toxicology's 43rd Annual Meeting. March 21-25. Baltimore, MD.

135. Santamaria, A., A. Li, F. Mowat, C. Cushing and **B.L. Finley**. 2004. Potential neurological effects of manganese exposure during welding: A "State-of-the-

Science" review. Abstract # 394. Presented at Society of Toxicology's 43rd Annual Meeting. March 21-25. Baltimore, MD.

136. Paustenbach, D. J., G. Brorby, and **B.L. Finley**. Environmental and Occupational Health Hazards Associated with the Presence of Asbestos in Brake Linings and Pads (1900 to Present). Society For Risk Analysis Annual Meeting. December 7-10, 2003. #T14.6. Baltimore, MD.

137. Connor, K.T. and **B.L. Finley**. 2003. A framework for evaluating relative potency data in the developments of toxicity equivalency factors (TEFs). Abstract # 664. Presented at Society of Toxicology's 42nd Annual Meeting. March 9-14. Salt Lake City, UT.

138. Cushing, C.A., K.C. Holicky, D.W. Pyatt, D. Staskal, **B.L. Finley**, D.J. Paustenbach and S.M. Hays. 2003. Estimated children's exposure to decabromodiphenyl oxide in the U.S. Abstract #1906. Presented at Society of Toxicology's 42nd Annual Meeting. March 9-14. Salt Lake City, UT.

139. Paustenbach, D.J., **B.L. Finley**, and A.K. Madl. Quantitating dose and risk: A case involving chromium in groundwater. Workshop on Recent Improvements in the Practice of Risk Assessment as Illustrated through Case Studies. 18th Annual International Conference on Contaminated Soils, Sediments, and Water. October 22, 2002. Amherst, MA.

140. **Finley, B.L.** and S.H. Su. 2002. Contaminated sediments and bioaccumulation criteria: procedures for evaluating dredging and open ocean disposal permits in the New York/New Jersey harbor. Presented at Proceedings of International Conference on Remediation of Contaminated Sediments. October 10-12, 2002. Venice, Italy.

141. Madl, A.K., **B.L. Finley**, J. Warmerdam, R. Richter and D.J. Paustenbach. 2001. Contributions of individual truck operations to ambient diesel particulate matter (DPM) concentrations: Implications for Risk Assessment and Management. Abstract #2063. Presented at Society of Toxicology's 40th Annual Meeting. March 25-29. San Francisco, CA.

142. Proctor, D.M., **B.L. Finley** and D.J. Paustenbach. 2001. Is hexavalent chromium carcinogenic via the oral route of exposure an evaluation of the state of the science and implications for drinking water pregulations? Abstract # 1499. Presented at Society of Toxicology's 40th Annual Meeting. March 25-29. San Francisco, CA.

143. **Finley, B.L.**, K. Fehling, J. Green and S.M. Hays. 2000. The presence of the Ah-receptor agonists in the diet: Implications for risk assessment and risk management. Presented at the International Dioxin Meeting 2000. Monterey, CA.

144. **Finley, B.L.**, K. Conner, J. Otani and P.K. Scott. 2000. Weighted PCDD/F and PCB REP distributions and their use in probabilistic risk assessment. Presented at the International Dioxin Meeting 2000. Monterey, CA.

145. Bernhardt, T., **B.L. Finley**, M. Goodman and K. Connor. 2000. An estrogen equivalents comparison of daily doses of bisphenol a from polycarbonate baby bottles vs. naturally-occurring compounds. Abstract #1201. Presented at Society of Toxicology's 39th Annual Meeting. March 19-23. Philadelphia, PA.

146. Connor, K., V. Craven, N. Wilson, T. Iannuzzi, D. Ludwig and **B.L. Finley**. 2000. A probabilistic human health risk assessment for the intake of polychlorinated biphenyls (PCBS) in ANGLERS OF THe Lower Fox River, Wisconsin. Abstract # 1173. Presented at Society of Toxicology's 39th Annual Meeting. March 19-23. Philadelphia, PA.

147. Exuzides, A. and **B.L. Finley**. 2000. A study of latex allergy using the national health and nutrition examination survey. Abstract # 590. Presented at Society of Toxicology's 39th Annual Meeting. March 19-23. Philadelphia, PA.

148. **Finley, B.L.**, D.J. Cher and S.M. Hays. 2000. Natural rubber latex allergy: A critical review. Abstract # 1160. Presented at Society of Toxicology's 39th Annual Meeting. March 19-23. Philadelphia, PA.

149. Proctor, D.M., J.R. Nethercott, M.M. Fredrick, **B.L. Finley** and D.J. Paustenbach. 1997. Assessing the potential for elicitation of allergic contact dermatitis in Cr(VI)-sensitized subjects following prolonged contact with Cr(VI) in solution. Abstract # 1051. Presented at Society of Toxicology's 36th Annual Meeting. March 9-13. Cincinnati, Ohio.

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